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RECURRENT INTRA-OCULAR HAEMORRHAGE IN YOUNG ADULTS (EALES' DISEASE)

Being an account of the Disease, a Summary of the Literature, a Report of Five Cases, and some Speculations as to the Aetiology

BY

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Summary of the Literature and Historical Remarks

ALTHOUGH it is usually stated that Eales first defined this group of cases, it appears that von Graefe recognized the group 25 years before Eales' first publication. In 1855 von Graefe[11] (quoted from Krauss[15]) mentioned the disease, describing periodical intra-ocular haemorrhages in young individuals who had often had previous epistaxis.

In 1880, Henry Eales[8] of Birmingham, published his first paper on "Cases of Retinal Haemorrhage Associated with Epistaxis and Constipation."

He described five cases of recurrent retinal and vitreous haemorrhage, all in young men, their ages ranging from 14 to 29 years. The fifth case differs from the others in many respects and is introduced "as a contrast." The other four were lads, the subjects of epistaxis and constipation. Eales described "symptoms of high arterial tension, such as slow pulse, and accentuation of the heart sounds." Two had traces of albumen in the urine, but no casts. In all the cases the left eye was mainly affected; in only one, was

the right eye also affected. He found no evidence of constitutional disease of any sort. He attributed the condition to a neurosis affecting the circulatory organs and digestive system, leading on the one hand to partial inhibition of the bowel movements, and to a vasomotor contraction of the vessels of the alimentary canal, causing dyspepsia and constipation, and on the other hand to a compensatory dilatation of the systemic capillaries, especially those of the head, and, in these cases, of the retina, with a tendency to rupture on the occurrence of any intensifying cause. He suggested that the limitation of the haemorrhages to the nose and the retina was due to a lack of counterpoising support in these situations.

Eales attributed the occurrence of the haemorrhages mainly in the left eye, to the facts that the left carotid artery arises more directly from the aortic arch than the right, and that the course of the left innominate vein is more lengthy and indirect than the right; these circumstances producing slightly greater capillary tension on the left.

He suggested that females are saved from this type of intra-ocular haemorrhage by menstruation.

In one of Eales' cases, vision in the left eye was completely lost through the occurrence of glaucoma.

Jonathan Hutchinson[14] in 1880, a few months after Eales' publication, described a case of primary intra-ocular haemorrhage in the left eye, followed after a long interval by haemorrhage into the right eye. The left eye was excised as the condition was complicated by glaucoma. The right eye was affected over two years later. There were repeated relapses of haemorrhage in the right eye, but glaucoma did not occur. On one occasion a retinal vessel actually ruptured while the eye was being examined. The left eye, after excision, showed haemorrhages in the vitreous, between the retina and the vitreous, in the ciliary processes and in the retina near the disc.

This case and one other, described by Hughes[13] in 1929 are the only two discovered in the literature in which it was possible to dissect an eye affected by this condition.

This (Hutchinson's) patient suffered from epistaxis and constipation; there was no sign of constitutional disease, but there was a strong family history of gout.

Hutchinson attached importance in these cases to "inheritance of the gouty state," and disagreed with Eales on the importance of constipation as a starting point.

In an appendix he described in less detail four other cases, in one of which there was a suspicion of syphilis.

In 1882 Eales[9] published another paper on "Primary Retinal Haemorrhages in Young Men." He suggested the designation

"Primary Recurrent Retinal Haemorrhage." He outlined again the characters of the disease and described lassitude and dyspepsia as occurring in all his cases; he did not support Hutchinson's view of the gouty diathesis as a cause.

No fresh cases were found in 12,000 patients treated at the Birmingham and Midland Eye Hospital in one year.

The retinal haemorrhages were described in more detail as large and round, or irregular; *not* flame shaped; and as a rule, confined to the periphery of the retina, near the veins. Vision only suffered in proportion to the vitreous opacity, which often cleared very rapidly between attacks. The ultimate results were large, whitish glistening patches in the periphery of the retina, and branched vitreous opacities. Small retinal detachments occurred sometimes.

Glaucoma occurred in one case.

Eales referred to three further cases, less fully investigated, which confirmed his previous views.

Nieden[19] (quoted from Krauss[15]) in 1882 reported that in 34,489 consecutive eye cases in 8 years 6 months, he found only six patients suffering from idiopathic recurrent vitreous haemorrhage. In three cases the second eye was not affected; in the other three, both eyes were affected alternately. He did not agree with Eales that the left eye was mainly affected, nor with Eales' argument to explain this. Of the nine eyes affected in his six cases, five were right and four left. There was no history of syphilis or tuberculosis; they were not constipated, but lacked appetite and energy. He believed the haemorrhage was from the choroidal vessels.

Mayweg[16] (quoted from Krauss[15]) in 1889, reported the case of a boy, aged 17 years, who lost his left eye as a result of intra-ocular haemorrhage and detachment of the retina. The right eye was subsequently affected by haemorrhage, which was stopped by ligature of the right common carotid artery.

Henry[12] in 1894, described a typical case in a young man, aged 19 years, of "effeminate type," who had recurrent intra-ocular haemorrhages. The right eye was affected, the left remaining normal. He suffered from headache, constipation and epistaxis. He had several recurrences in four months, but in one year the vision had recovered to 6/8. The retinal haemorrhages were chiefly peripheral; the vitreous haemorrhages cleared quickly. Glistening white patches remained in the retina.

Krauss[15] in 1908 reported a case in a young man, aged 23 years. The right eye alone was affected; he suffered from constipation, but never from epistaxis. Recurrent vitreous haemorrhages were frequently preceded by violent headaches. Rapid recovery of vision took place between attacks. Areas of "choroiditis" and vitreous veils were left by the disease.

Krauss suggested the existence of a condition of local disease or weakness of the retinal or choroidal vessels, permitting rupture when other conditions were favourable. He stated that in individuals liable to epistaxis, the vessels of the nasal mucosa are near the surface, possibly with degenerated walls, and bleed easily. He suggested that such a condition might exist in the choroidal capillaries in the subjects of this disease. Any exciting cause, producing change in blood pressure (as indicated by severe headaches preceding the attacks in the above case) might then cause rupture of the weak vessels.

In this paper, Krauss quotes von Graefe[11] and Eales;[8] also Nieden[19] and Mayweg.[16]

Noll[20] (quoted from Finnoff[10]) in 1908, first suggested that the disease might be due to a tuberculous process.

Axenfeld[3] (quoted from Finnoff[10]) in 1910 also suggested that these cases were often due to tuberculosis although no apparent tuberculous changes were present elsewhere in the eye; the lesions were due either to the bacilli themselves or to their toxins. The patients were frequently robust. Tuberculin was used for diagnosis and treatment, and improvement frequently followed its use.

Davis[7] in 1912 reported a case in a young man, aged 22 years, the subject of dyspepsia but not of constipation. He had recurrent haemorrhages into the retina and vitreous of each eye, which began in the right. Within a year he lost the vision of the right eye almost completely, due to irreparable damage done by the haemorrhages; and the vision of the left eye was very seriously impaired, due to retinitis proliferans, and detachment of the retina. Davis observed "dense broad white bands" round the veins of the retina. There were some patches of white exudate between the veins.

In this case there were signs of kidney trouble at one time during the disease, shown by slight albuminuria, with many casts (red cells, granular and epithelial) in the urine; this however was only temporary, and the urine was again normal within three weeks.

Old tuberculin was used subcutaneously for diagnosis and treatment. General and local eye reactions resulted from the injection of 0·2 mgm. of old tuberculin, flushing of the sclera of each eye was observed.

Injections of human serum were tried without success.

Vitreous opacities, retinitis proliferans, and retinal detachment resulted from the disease. Davis concluded that tuberculosis was a definite aetiological factor, as also was indigestion and auto-intoxication. He considered that the haemorrhages arose from the retinal and not the choroidal vessels.

He favoured tuberculin and general hygienic management in the treatment.

Harrison Butler,[5] in a discussion on the subject in 1912, quoted Axenfeld.[3] He regarded a local reaction in the eye, following the injection of old tuberculin (subcutaneously) as conclusive evidence of tuberculous aetiology. He thought it reasonable that this condition might be due to local or remote tuberculosis.

Bennett[4] in 1913 reported the case of a woman, aged 23 years, with retinitis proliferans in the left eye and incipient retinitis proliferans and some small retinal haemorrhages in the right eye. He regarded it as a case belonging to this group although he makes no actual mention of occurrence, of epistaxis, or of constipation. The right vision was reduced to 6/24, and the left to 6/60. Treatment was of no avail until thyroid extract was given, when the eye condition improved greatly, and the right vision became 6/6 partly and the left 6/18.

Ziegler,[23] in 1912, also advocated the use of thyroid extract in treatment.

Zentmayer,[22] in 1920, outlined the disease as described by Eales. He quoted four cases of his own; in all, the Wassermann reaction was negative; in two the von Pirquet test was positive. Coagulation of the blood was delayed in two cases.

In view of the constant age incidence, the frequency with which males are affected, the physical condition of lassitude, the cold hands, and asthenia, and the benefit derived from the use of thyroid extract, Zentmayer suggested a disturbance in activity of the ductless glands as a factor in the disease. Possibly the adrenals were deficient in activity causing lack of tone in the venules and predisposing to haemorrhage.

Zentmayer did not wholly reject tuberculosis as a cause but thought some other factor must be found.

Aubineau[2] in 1920 reported five cases of intra-ocular haemorrhage in adolescents (apparently falling into this group). These cases were in hospital at the same time and were treated by the deliberate induction of anaphylactic shock by the injection of serum at suitable intervals. The injections were given under the skin and beneath the conjunctiva. In three cases no satisfactory general reaction was obtained. One case appeared to be definitely improved by each general reaction. (see also Candiotti.[6])

Finnoff[10] in 1922 reported five male cases; three of these were over the age of 30 years; one, aged 42 years, was the subject of pulmonary tuberculosis. Another had some central nervous disorder, with paraplegia and loss of sphincter control. It does not seem quite clear that these cases should be included in the group.

Finnoff concluded that this was not a specific disease and that tuberculosis of the retinal vessels, especially the veins, was one common aetiological factor. A focal reaction to the tuberculin test proved this.

Adams,[1] in a discussion on the significance of retinal haemorrhages in 1922 did not agree that tuberculosis was an aetiological factor.

Candiotti[6] in 1922, described the case of a girl, aged 17 years, the subject of pulmonary tuberculosis and of albuminuria, who had recurrent vitreous haemorrhages. Anaphylactic reactions were deliberately produced by the injection of various sera, with apparent benefit to the eyes. However a severe recrudescence of the pulmonary disease also followed the treatment.

Foster Moore[17] states in his book "Medical Ophthalmology" that there is no satisfactory evidence that the condition is due either to tuberculous infection or is associated with functional albuminuria.

He refers to the case of a woman in whom recurrences of the haemorrhage tended to occur at the menstrual periods and who had already lost the sight of one eye. The ovaries were removed in the hope of arresting the haemorrhage; the periods ceased but the retinal haemorrhages persisted, and total blindness resulted.

Hughes[13] in 1929, reported a case in a youth, whose eyesight had been previously passed in a Board of Trade test.

Both eyes were affected; recurrent haemorrhages occurred during a period of about 2 years. The left eye was chiefly affected, became blind and glaucomatous. Hughes removed this eye. The retina was completely detached by haemorrhagic subretinal exudate. There were many areas of haemorrhage in the choroid. There is no mention of microscopic examination. The general health was good. Nearly 3 years after the onset, the right vision (corrected) was 6/6.

Young[21] in 1930 reported three cases of recurrent haemorrhage into the retina and vitreous in adolescents. One case showed deficiency in blood calcium, and seemed to benefit from the administration of calcium.

From this survey of the literature it is evident that many theories have been put forward to account for the occurrence of the haemorrhages in this group of cases, but no theory has been proved beyond doubt.

Many forms of treatment have been tried but no consistent success has been reported. Our ignorance of the cause of the disease must be the greatest obstacle to the discovery of effective methods of prevention and treatment.

General Account of the Disease

Incidence and Aetiology.—The disease is very rare. Nieden[19] found only six in 34,489 consecutive eye cases. Aubineau[2] found eight in 25,000. It occurs in young men between the ages of 15

and 25 years. Women are not immune, but are affected much more rarely than men. In the cases reported in the literature the ratio is about nine to one.

Although one eye alone is first affected, the other one usually shows signs of the disease or is involved later. Eales[8] found that the left eye was more often affected, but the cases reported in the literature show that both eyes are affected in the majority and cases in which one eye alone is affected are divided evenly between right and left.

The aetiology of the disease is still unknown; various theories have been put forward. Eales[8] suggested a neurosis affecting the alimentary and circulatory systems. Hutchinson[14] suggested an inherited gouty state. Delayed blood coagulation, deficient blood calcium, haemophilia, local weakness of the retinal or choroidal vessels, endocrine disorder, the effects of the tubercle bacillus or its toxins, and helminthiasis have at various times been suggested as the cause of the haemorrhages.

Precipitating causes of haemorrhage in the subjects of the disease are the recumbent position, coughing, sneezing and any sudden strain.

Morbid Anatomy and Pathology.—Very little is known of the morbid anatomy of the disease, as the necessity to remove an affected eye rarely arises. In only two cases in the literature has an affected eye been dissected; in each case the eye was removed because of glaucoma. The first case was described by Hutchinson;[14] haemorrhages were found in the retina near the disc, in the ciliary processes, between the retina and the vitreous, and in the vitreous. The second case was described by Hughes,[13] the eye removed showed the retina completely detached by haemorrhagic subretinal exudate and the choroid engorged with many areas of haemorrhage in it. No mention is made, in either case, of microscopic examination.

Clinical Features; Signs and Symptoms.—The subjects of Eales' disease are as a rule spare youths, fairly well nourished and apparently in good health, but they are very frequently lacking in spirits and energy. They have a poor appetite and often suffer from discomfort and flatulence after food. Constipation is common but not always present. They very often suffer or have suffered from attacks of epistaxis. Apart from these conditions there is no sign of constitutional disease; syphilis, haemophilia, anaemia, nephritis, heart disease and diabetes are not found. The blood is usually normal to all tests. The pulse is sometimes slow, the blood pressure low or normal. Functional albuminuria has occasionally been found. Some cases have suffered from pulmonary or lymphatic tuberculosis, but the relation of tuberculosis to the intra-ocular haemorrhages has not been conclusively proved.

The disease is characterised by the sudden occurrence of intra-ocular haemorrhage, for no obvious reason, producing dimness of vision. One eye is usually primarily affected and the patient may only notice this accidentally when the other eye happens to be covered. There is no pain or change in the external appearance of the eye.

The patient comes to the doctor and complains that a short time previously he noticed a mist, or floating shadows, before one eye; he may even describe a red glare, obscuring the vision of one eye. Frequently he takes little notice of the first attack, as he is but slightly inconvenienced by it; but the recurrence of his symptoms within a few days or weeks ultimately causes him alarm, and he seeks advice.

On examining the affected eye with the ophthalmoscope, it is found that the cause of the loss of vision is intra-ocular haemorrhage. The haemorrhages occur in the retina, and may or may not break through into the vitreous humour. Unless the macula itself is affected, which is uncommon, loss of vision seems to be in proportion to the degree of opacity resulting from haemorrhage into the vitreous. Quite extensive haemorrhages in the retina may not cause the patient to notice anything wrong.

It is often impossible at first to discover the full extent and characters of the retinal haemorrhages on account of the blood present in the vitreous. When the vitreous has cleared and the fundus can be examined, the retinal haemorrhages are found to be mainly peripheral, often situated near or beneath the retinal veins. They are less commonly seen near the disc or macula. The haemorrhages are usually large and irregular, often with serrated edges. Flame-shaped, striated, and punctate haemorrhages are seen, but not commonly. The haemorrhages show brown discolouration as they degenerate.

Semi-circular subhyaloid haemorrhages are sometimes seen.

White patches of exudate have been described, but are not common. A star figure at the macula does not appear. White bands along the course of the retinal vessels (usually the veins) are sometimes present at the periphery of the fundus.

Pigmentary disturbance is not seen, except at the site of old haemorrhages.

The retinal veins are usually dark, full and tortuous.

When the patient is first examined the vitreous chamber may be so full of blood that the fundus cannot be seen except at the extreme periphery. The vitreous then appears quite black when the ophthalmoscope is used, a dull red glare being seen by oblique illumination, or there may be numerous branched opacities in the vitreous, having the appearance of floating black cobwebs, between which a red reflex from the fundus is seen.

The conjunctiva, cornea, sclera, anterior chamber, iris, pupil, and lens are normal, unless such complications as glaucoma and iritis set in.

It is a curious characteristic of the disease that the blood in the vitreous is absorbed with surprising rapidity, the remaining opacities sink to the bottom of the vitreous chamber, leaving the upper part of the vitreous comparatively clear. Thus a vitreous haemorrhage may reduce the visual acuity suddenly to perception of light only or to counting fingers, but in two or three weeks vision may be restored almost to normal.

The course of the disease is characterised by recurrences of haemorrhage, which occur over a period of months or years. The second eye is very frequently affected. After the first attack, although vision may have been very much reduced, it may recover almost to normal within a few weeks. Then, suddenly, fresh haemorrhages occur, sometimes quite spontaneously, or as a result of a violent cough or sneeze, and vision is again greatly obscured by a large vitreous or subhyaloid haemorrhage. In spite of all treatment, recurrences may occur for several years with depressing regularity. They are sometimes accompanied by deterioration in the general health, recurrence of dyspepsia, constipation, epistaxis and bradycardia, and finally, again for no obvious reason, the haemorrhages cease.

The final condition of the eye, or eyes affected, after the haemorrhages have ceased, depends on the magnitude and number of the haemorrhages and the resultant damage to the eye, the extent to which they are absorbed and the presence or absence of complications.

The vitreous haemorrhages may clear almost completely, but as a rule some opacity remains. There may be a diffuse haze, or there may be branched floating opacities, attached in places to the retina. The vitreous opacities often sink to the bottom of the vitreous chamber. Subhyaloid haemorrhages are usually absorbed completely.

The retinal haemorrhages undergo gradual absorption; they may leave behind them pigmented patches or glistening white areas of atrophy in the retina. Veil-like opacities may come forward from the retina into the vitreous, carrying with them delicate newly-formed blood vessels.

Complications.—Retinitis proliferans is probably the commonest complication and is often described in the literature. White bands of connective tissue form on the retina and in the vitreous.

Detachment of the retina is fairly common and is a great menace to vision after the haemorrhages have ceased. It is associated with retinitis proliferans, the contracting bands of connective tissue pulling on the retina and detaching it.

Chronic glaucoma and iritis are uncommon complications. Glaucoma has been reported in two cases in the literature and iritis in one.

Prognosis.—The disease is confined to the eye and is not necessarily associated with any constitutional disorder. There is therefore no danger to life.

The immediate prognosis as regards vision is bad. Both eyes are usually affected, one less severely than the other.

Recurrences are likely to occur for months or years, with greater or less obscuration of vision. Between the attacks, vision may return almost to normal.

The ultimate prognosis, as mentioned before, depends on the magnitude of the haemorrhages and their damage to the retina and vitreous; the duration of the disease and the occurrence of complications. A number of large haemorrhages may do irreparable damage to the eye, producing extensive scars in the retina and permanent opacities in the vitreous. On the other hand, very large vitreous haemorrhages, if not too frequently repeated, may be almost completely absorbed and good vision recovered. However, even if this occurs there is grave danger that the retina may be detached by the growth and contraction of fibrous bands attached to it, and the eye is almost certain to become blind in time. The eye may be lost through the occurrence of chronic glaucoma while the disease is still progressing, but this is uncommon.

Diagnosis and Differential Diagnosis.—Eales' disease is very rare, and before the diagnosis is made in a case of recurrent intra-ocular haemorrhages in a young adult, it is necessary to exclude a number of other conditions in which such haemorrhages may occur. Such conditions are nephritis, diabetes, the anaemias, leukaemias, purpuras, and syphilis. The stools should be carefully searched for the ova and segments of parasitic worms, since retinal haemorrhages are frequent in cases of bothriocephalus anaemia. However, anaemia is not the rule in Eales' disease, whereas it is always present in cases of retinal haemorrhage caused by worms in the alimentary canal.

Subcutaneous injections of Old tuberculin have been used for diagnosis; a focal reaction in the eye, with flushing of the bulbar conjunctiva being taken as evidence of tuberculous aetiology.

Trauma should, of course, be excluded as a cause and the possibility of malignant intra-ocular growths should always be borne in mind.

Treatment.—No consistently successful method of treatment has yet been discovered.

(a) *Local Treatment.*—This consists of giving the eyes complete rest so long as the haemorrhages continue. Reading should be

forbidden and dark glasses ordered. It is probably wise to keep the eyes under the influence of atropine while the disease is active.

Both hot and cold applications have been used with the object of promoting absorption of the blood. Subconjunctival injections of dionine and saline have been tried, without great success.

(b) *General Treatment.*—The patient should not be allowed to work while the disease is progressing. It is advisable to give complete rest in bed while the general investigation is being done. At the same time it is possible to judge whether complete rest is beneficial to the patient. In most cases it seems better to allow and encourage reasonable exercise; confinement to bed tends to increase mental depression and lassitude, and low spirits are common characteristics of the disease. Moderate exercise will increase the appetite which is often poor and possibly aid the sluggish digestion. Moreover, it is usually found that the haemorrhages occur just as frequently when the patient is in bed as when he is up and about. On the other hand, strain of any sort, physical or mental, should be avoided. A "change of air" is probably beneficial if the patient becomes depressed by the tedious course of his disease.

General hygienic measures should be adopted; the constipation corrected by the use of mild laxatives and an occasional saline purge if necessary. Every effort should be made to improve the appetite and digestion by suitable diet and the use of bitter tonics.

Thus far and no farther, can one go in the treatment with any confidence of definitely doing good. Various other forms of medical treatment have been tried. Potassium iodide has been given, in the hope of promoting absorption of the haemorrhages. Thyroid extract has been administered, and several writers have found it beneficial.[4, 22, 23.]

When the blood calcium is reduced, calcium and parathyroid extract may be tried;[21] indeed this treatment has been employed even when the blood calcium was normal, as it usually is.

Injections of human serum have been used;[7] and apparent success has been reported from the deliberate production of anaphylactic shock by the injection of anti-diphtheritic serum.[2, 6.]

The injection of Old tuberculin in cases giving a local or general reaction to the intradermal or subcutaneous test may be tried.[7, 10.]

The galvanic current, applied locally to the eye, has been used.

Report of Five Cases (for full notes, see Appendix)

The cases investigated have all been treated as in-patients in the Ophthalmic Ward at St. Bartholomew's Hospital, London. One of the cases was admitted and treated while the writer was

House Surgeon to the Ophthalmic Department. (This case is still under observation, as the disease remains active.) The records of the ophthalmic cases for the past 7 years 6 months (*i.e.*, from December, 1923 to May, 1931, inclusive) were examined and the notes of the four other cases discovered. Of these four cases, three have attended hospital as out-patients since their discharge and further notes have been made of their condition from time to time. Of the three who have attended as out-patients, two were recently re-examined by the writer, the third could not be traced, and has not been seen at the hospital for nearly four years.

The fourth patient, who has never attended as an out-patient since his discharge, has been examined at intervals by Mr. Arthur Cooke, at Addenbrooke's Hospital, Cambridge. I am greatly indebted to Mr. Cooke for his kindness in giving me further notes of this case.

The following are the important facts about the five cases collected:—Five cases of Eales' disease were admitted to the Ophthalmic Ward in 7 years 6 months, during which time 2,559 eye cases were admitted to the hospital. (1,255 males and 1,304 females.) These figures, however, are practically valueless, since all five cases were sent to the hospital from considerable distances in the country, as cases of exceptional interest, for investigation.

The age of the patients at the onset of the disease was between 17 and 25 years.

Four cases were males, one female.

Two patients were definitely lacking in spirits and energy.

In two there was a history of epistaxis; in one other case epistaxis occurred during the course of the disease, although it had never happened before. In one case there was no epistaxis; and in one it is not mentioned. In no case was there any other tendency towards prolonged bleeding or bruising. In *no* case was constipation present. Discomfort and flatulence after food were complained of in two cases. Frontal headache was a feature in two cases, and was present occasionally in a third. Bradycardia was not present except in one case, in which the pulse frequency was usually about 60 in the mornings but more rapid in the evenings. The blood pressure was within normal limits in each case.

In no case was any sign of tuberculosis found. An intradermal tuberculin test was done in the worst and most recent case, and was negative. In one case only was there a family history of tuberculosis, a sister being suspected of pulmonary infection.

There was no suspicion of syphilis, congenital or acquired, in any case. The blood Wassermann and Sigma reactions were negative in each case.

Urine examinations and the blood coagulation time were normal in each case.

Blood counts were done in four cases and were found normal.

The blood platelets, blood calcium and bleeding times were estimated in two cases and also found normal.

No case had had any previous trouble with the eyes.

In all five cases both eyes were involved to a greater or less extent; in two cases, in the less affected eye, one small retinal haemorrhage was the only discoverable sign of disease.

In one case (the worst) both eyes were affected equally; in another case the left eye was mainly affected, and in the other three the right eye was mainly affected.

Vitreous haemorrhage occurred in all cases; in three, only in the eye chiefly affected, and in the other two, in both eyes.

In one case the vitreous haemorrhage was slight and the vision after recovery was good. In the other four cases the vitreous haemorrhages were large and the vision after recovery very poor. (In the case in which the disease is still active, the final result is of course not known, but it seems certain that vision will be very poor.)

The retinal haemorrhages in three of the cases were mainly peripheral; in two the haemorrhages were situated both near the disc and at the periphery. The haemorrhages were in two cases large and irregular; in three small and circumscribed. Flame-shaped and striated haemorrhages were not seen.

The retinal veins were full, dark and tortuous in one case. In another, they were surrounded by thin white bands near the periphery. Apart from this the retinal vessels were normal.

In each of three cases, a patch of white exudate was seen in the retina.

The total period during which the haemorrhages took place, varied from six months to four years. From three to five recurrences of haemorrhage occurred as a rule; except in the case of the patient most recently seen whose disease is still progressing and who has already had repeated haemorrhages in both eyes. One patient, whose disease lasted two years, is known to have been free from haemorrhages for three years. Another, who had recurrences for four years, has now been free for two years.

In three cases in which large vitreous haemorrhages occurred, permanent vitreous opacity has resulted; vision was reduced in the affected eye to 6/60 in one case and to even less in the other two patients.

In the case in which the vitreous haemorrhage was slight the vision is now 6/6 in both eyes, three years after the last haemorrhage. What opacity is left in the vitreous, sinks to the bottom of the chamber, causing no disturbance of vision. A few veil-like

opacities with delicate new vessels project from the retina into the vitreous of the affected eye in this case.

Two other patients are now usefully employed at work which requires good eyesight; they rely on the less affected eye.

Complications, such as detached retina, chronic glaucoma, and iritis have not occurred so far in any case.

Discussion

The results of the investigation of these five cases, in the main, agree with the facts already established about the disease.

That the male sex is chiefly affected is confirmed, but the female sex is not immune. The age incidence, 17 to 25 years, is also in agreement with previous findings. The general characters of the disease in the cases investigated are the same as those previously described, except for a few minor points of difference. Constipation, for instance, was not a feature of these cases. The prognosis as regards vision in the eye chiefly affected is perhaps not so good as is indicated by some writers; gross vitreous opacities remained in at least three cases. No sign of tuberculosis was found in any of the patients.

The chief matter of interest in Eales' disease is its aetiology, which is still unknown.

Eales' own theory as to the importance of constipation and intestinal disturbances as a starting point, does not seem to have received much support. It is difficult to believe that a neurosis producing partial inhibition of the bowel movements and vaso-motor contraction of the blood vessels of the alimentary canal could produce a compensatory dilatation of the systemic capillaries sufficient to cause rupture and haemorrhage in the eye, on the occurrence of any slight intensifying cause.

Deficiency in blood calcium, prolonged bleeding time and delayed blood coagulation, seem to be ruled out in almost every case in which these points have been investigated.

Zentmayer's theory[22] that there might be disturbance of ductless gland activity, does not seem to have been tested sufficiently in actual practice. The fact that thyroid extract benefits some cases gives some support to the theory. Zentmayer suggested that adrenal deficiency might cause lack of tone in the venules and and predispose to haemorrhage. It would be interesting to try the effect on the disease, of injections of adrenalin or of ephedrin given by mouth.

The theory that tuberculosis is an aetiological factor lacks any concrete evidence in its support, and although several writers have

favoured the theory, none has offered any pathological explanation of the process whereby the tubercle bacillus, either by its local presence in the eye or by the toxaemia produced by its remote presence in the body, could produce the haemorrhages characteristic of this disease.

Tuberculous toxin does not produce haemorrhages in other parts of the body.

In any condition characterized by haemorrhage, one must assume that there is an abnormality either of the blood vesels, or of the blood itself, or of both. In Eales' disease, there is so far no pathological evidence of either. Krauss[15] has suggested a condition of weakness or local disease of the choroidal or retinal vessels permitting rupture when other conditions are favourable. If this is the case, why does the condition start so abruptly in adolescence, only to cease, also abruptly, after some months or years? If an abnormality of the blood causes the haemorrhages, why does it affect only the eye and the nose? Is it because there is lack of counterpoising support in these situations? (as suggested by Eales), or do haemorrhages occur, unrecognised, in other parts of the body?

It appears most likely that there is some abnormality of the blood, as yet unrecognized by pathological tests: it might be a deficiency of some normal constituent, producing reduced viscosity or altered osmotic pressure; or it might be the presence of some abnormal toxic constituent, absorbed from the bowel. The general condition of lassitude and low spirits, so common in the subjects of the disease, supports this view. Such a condition of the blood might conceivably have a harmful effect on the venules and capillaries of the body, so that they allow diapedesis to occur or even actual rupture. Foster Moore[18] believes that most retinal haemorrhages (not specially in Eales' disease) are brought about by diapedesis. Leakage of blood from the vessels in the eye would soon produce symptoms and easily recognized signs. It may be that the haemorrhages occur only in the eye and the nose because of a lack of supporting tissue around the vessels in these situations.

Another possibility is that the disease might be a manifestation of vitamin deficiency, allied to scurvy. This idea was suggested by the frequency with which reported cases of Eales' disease have occurred in youths in some way connected with the sea; this circumstance can, of course, have no bearing on the matter in these days. In scurvy, epistaxis and constipation are common, conjunctival haemorrhages not uncommon, but retinal haemorrhages are quite unusual. There is nothing else suggestive of scurvy in Eales' disease.

Conclusions

1. The disease is rare, affecting young men between the ages of 17 and 25 years. The female sex is not immune.

2. Epistaxis is common, but does not always occur.

3. Constipation and dyspepsia are not invariably present.

4. There is no general constitutional disease, but a condition of lowered vitality is often present.

5. One eye is chiefly affected, either right or left, but the second eye is almost always affected to a greater or less degree.

6. The retinal haemorrhages are chiefly peripheral.

7. Vitreous haemorrhage is the rule, in the eye chiefly affected.

8. The prognosis as regards vision in an affected eye, depends mainly on the extent of the vitreous haemorrhages: large and recurrent haemorrhages tend to produce permanent opacities with much impairment of vision.

9. There is no satisfactory evidence that tuberculosis, either local or remote, is a cause.

10. The disease is probably due to deficiency in some blood constituent, or to the presence of some toxic product, causing damage to the endothelium of the smaller branches of the retinal vessels, with resultant diapedesis.

11. The occurrence of the haemorrhages chiefly in the eye may be due to the lack of supporting connective tissue around the retinal vessels.

Appendix

NOTES OF CASES

CASE I.—J. R., male, hatter. First attended the out-patient department on April 20, 1926, then aged 25 years. Admitted to hospital on April 23, 1926.

History: Eight days previously had noticed a black blur over the right eye. Four days previously, black blur changed to a green mist, which since then had been clearing. No previous trouble with the eyes; no bruising; epistaxis when younger; no jaundice; no constipation; no dyspepsia.

Examination: General physical examination revealed nothing abnormal. Blood pressure: 130 systolic, 80 diastolic.

Right eye: External examination, normal. Vision 6/12. Vitreous: blood was present, but the opacity did not greatly obscure the fundus. Fundus: two or three large irregular haemorrhages were seen in the retina near the disc, at its lower and nasal margins; two other elongated irregular haemorrhages were seen, one around the lower temporal vein and the other near the lower nasal vein. The haemorrhages showed brownish discolouration. A brownish elongated patch was seen on the nasal side of the disc, apparently the remains of an older haemorrhage. An elongated haemorrhage with brownish discolouration at its centre was seen directly above the disc, about two disc-diameters from it. The vessels, optic disc and macula were normal.

Left eye: Normal, except one small haemorrhage seen in the lower temporal quadrant of the retina. Vision 6/6. Blood coagulation time: normal. Wassermann and Sigma reactions: negative.

The patient was discharged after eight days in hospital. The right vision had improved to 6/9. Potassium iodide, grains v, three times a day, were given; also mild aperients. A fresh retinal haemorrhage appeared on May 21, 1926. On July 19, 1928, more than two years after the onset, there was a recurrence of symptoms, but no detailed note of ophthalmoscopic appearances was made. Since that time, no recurrence has occurred. The patient was examined again on April 21, 1931. He had no complaint and was following his occupation without difficulty. He suffered from indigestion but not from constipation. Vision was 6/6 in each eye. Greenish-grey opacities were seen in the lower part of the right vitreous chamber and greyish films projected into the vitreous from the retina; one of these veils was accompanied by delicate blood vessels. No fresh haemorrhages were seen, but a pigmented patch remained at the site of one of the early haemorrhages. The left eye was normal.

CASE II.—J. H., male, in "Naval Service." Admitted to ophthalmic ward on April 20, 1927, then aged 19 years.

History: One year previously, loss of vision in right eye; small specks in line of vision; clear in two weeks. Ten months previously, following excitement, a number of black specks appeared before the right eye. Admitted to Naval Hospital and treated by complete rest in bed. Very slight improvement; could only count fingers (with right eye) at this time. Left eye unaffected. Appetite and digestion good; no constipation; had only had one attack of epistaxis in his life; no headaches; no history suggesting syphilis or tuberculosis.

Examination: A healthy looking youth; general physical examination revealed nothing abnormal. Urine, normal; blood pressure: 108 systolic, 65 diastolic.

Right eye: External examination, normal. Vision 6/12. Vitreous: cloudy, with floating opacities (blood). Fundus: seen indistinctly; no visible haemorrhages in retina; disc, normal.

Left eye: External examination, normal. Vision 6/12. Vitreous: clear. Retina: several small haemorrhages in the temporal half and one patch of white exudate with a central pigmented area. Disc and macula normal. Blood count and coagulation time: normal. Wassermann and Sigma reactions: negative.

Treatment: Full diet: calcium lactate, gr. x, three times a day, and tab. parathyroid, gr. $\frac{1}{3}$, at night.

After three weeks in hospital the patient was discharged, not improved. He was examined again on May 22, 1927; there had been a recurrence of haemorrhage in the right eye and vision was reduced to finger counting; the right vitreous was completely opaque with blood. The left eye was as before, the vision being improved from 6/12 to 6/6 by correction of refractive error. On June 10, 1927, no improvement had taken place. Since this time the patient has not been traced.

CASE III.—W. M., male, assistant store-keeper. Admitted to ophthalmic ward on July 10. 1929, then aged 21 years.

History: Sent to the hospital by Mr. Arthur Cooke, of Cambridge, with the following notes:—Four years previously: while working as a stone mason, had a right vitreous haemorrhage; right vision reduced to finger counting. Three years previously: fresh effusion of blood in front of right retina. Two years previously: large floating opacities in both vitreous chambers. Five months previously: haemorrhage in left vitreous and retina; admitted to Addenbrooke's Hospital, Cambridge. under the care of Mr. Arthur Cooke. Blood count showed over 6 million red cells per cubic millimetre. Wassermann reaction: negative. Blood coagulation time: normal. Vision improved slightly to 6/60 (right) and 6/18 (left). One month previously: left vision reduced to perception of hand movements. Appetite good; digestion good; no constipation (epistaxis not mentioned). No history suggestive of tuberculosis or syphilis. A sister had been suspected of phthisis.

Examination: A healthy looking youth. General physical examination revealed nothing abnormal. Urine, normal; blood pressure: 124 systolic, 82 diastolic.

Right eye: External examination, normal. Vision 6/60. Vitreous: contained black branched opacities and many floating specks. Fundus; disc normal; several

small dark haemorrhages in retina on the temporal side, near an artery; on the nasal side was an oval white patch of exudate crossed by an artery.

Left eye: External examination, normal. Vision 6/12. Vitreous: contained many black opacities. Fundus: disc normal; above the disc was a large white patch with small haemorrhages in it; many small haemorrhages seen near the arteries; below the disc, a grey pigmented patch. Blood count and coagulation time: normal.

The patient was discharged after ten days in hospital; further notes supplied by Mr. Arthur Cooke stated:—"The patient has been well and at work since discharge; has complained of headache; no recurrence of haemorrhage. March 25, 1931, vision with glasses was barely 6/60 right and 6/12 left. In the right fundus, two broad white streaks, with small patches of pigment were visible in the temporal half, beyond the macula. The disc was normal. No marked opacities in the vitreous. In the left eye, vitreous opacities were well marked; the disc was pale with rather vague margins; a few faint pigmented patches were seen around the disc."

CASE IV.—A. A., female, clerk. Admitted on January 24, 1930, then aged 25 years.

History: Three weeks previously: woke up one morning to find the left vision misty; the condition had remained ever since. Right vision normal. No headaches; no previous eye trouble; no dyspepsia: no constipation; no epistaxis. Had always been healthy. No history suggestive of tuberculosis or syphilis.

Examination: Healthy looking. General physical examination revealed nothing abnormal. Urine, normal; blood pressure: 130 systolic, 90 diastolic.

Right eye: External examination, normal. Vision 6/6. Vitreous: clear. Fundus: normal, except one small retinal haemorrhage discovered at the periphery at "6 o'clock."

Left eye: External examination, normal. Vision, less than 6/60. Vitreous: diffuse central opacity, with several smaller floating opacities. Fundus; only the periphery visible; a few white patches and many small haemorrhages, especially at 3 and 6 o'clock. Blood count, platelet count, bleeding time, coagulation time and blood calcium all normal. Wassermann and Sigma reactions: negative.

The patient was discharged after two weeks in hospital. She was examined again on March 7, 1930. The vitreous opacities (left) had sunk to the bottom of the chamber and a good deal of absorption had taken place. Two large irregular haemorrhages were visible in the retina, near the disc. The patient was sent for again and examined on April 21, 1931. She reported that the left eye was worse, one or two recurrences of symptoms having occurred after she was last seen. No change had taken place for several months. The right eye was normal so far as she could tell, and she was following her occupation without difficulty. General health, very good. The right visual acuity was 6/5 partly. The left visual acuity was reduced to perception of hand movements. The right fundus was normal except for some fine sheathing of the finer vessels, which was seen at the periphery, especially below. The left vitreous appeared to be full of blood; indirect illumination showing a dull red opacity behind the lens; the fundus was invisible.

CASE V.—G. J., male, dockyard worker. Sent to St. Bartholomew's Hospital, London, by Dr. Heath, of Rochester, Kent, who had observed "every variety" of retinal haemorrhage in both eyes and marked alteration in the calibre of the retinal vessels. Admitted on March 11, 1931, then aged 19 years.

History: Five weeks previously: vision became misty in both eyes, left worse than right; this became slowly worse. Admitted to Naval Hospital, Chatham, and then transferred to St. Bartholomew's Hospital, Rochester, for investigation. The vision improved slightly. Sent to St. Bartholomew's Hospital, London, for admission under the care of Mr. Foster Moore. No history of injury or pain; two months previously, eyesight had been tested; vision then 6/6 right and left. Appetite good; troublesome dyspepsia and distension after meals; no constipation; no epistaxis; no bruising or prolonged bleeding. No history suggestive of syphilis or tuberculosis.

Examination: Healthy looking youth, but low in spirits and lacking in energy. General physical examination revealed nothing abnormal. Urine, normal; blood pressure: 110 systolic, 70 diastolic.

Right eye: External examination, normal. Vision 6/36. Vitreous: contained very many dark floating opacities; the bottom of the chamber was full of these.

Fundus only visible at the extreme periphery, above and to the nasal side; large irregular retinal haemorrhages, with serrated edges were seen in these situations.

Left eye: External examination, normal. Vision 6/9. Vitreous: Full of floating opacities, appearing to hang down from above; the lower part of the chamber was quite black. Fundus only visible at the periphery above, and to the temporal and nasal sides; a large irregular retinal haemorrhage visible on the temporal side.

March 13, 1931.—Vitreous opacities had cleared somewhat; various and extensive haemorrhages visible at periphery of retinae. March 31.—Recurrence of haemorrhage in both eyes, in spite of treatment and rest in bed. Vision 6/60 right, 6/24 left. April 4.—Vitreous humour even more opaque, especially left. April 6.—Practically no red reflex from left fundus, owing to increased vitreous opacity. April 11.—Right vision, 3/60; left vison, perception of hand movements only. April 17.—First attack of epistaxis; this occurred spontaneously while in bed. Second attack occurred two days later. April 28.—Right fundus more clearly visible; large irregular retinal haemorrhages noted. Left vitreous full of blood. May 1.—Right vitreous more opaque again. The disease is still progressing in both eyes.

Pathological tests: Blood count, blood platelets, bleeding and coagulation times and blood calcium, all normal. Wassermann and Sigma reactions, negative. Intradermal tuberculin test gave no local or general reaction.

Treatment adopted: Full diet; complete rest in bed at first, until it was seen that this had no beneficial effect; the discomfort after meals soon disappeared. Potassium iodide by mouth was tried without success for three weeks. Calcium lactate and parathyroid were then given but discontinued when it was discovered that the blood calcium was normal. Thyroid sicca was then given by mouth in doses up to gr. i, t.d.s.

I am indebted to Mr. Foster Moore for his kindness in allowing me to use the notes of these cases.

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METASTATIC CARCINOMA OF THE CHOROID. A REPORT OF TWO CASES WHERE THE PRIMARY NEOPLASM WAS IN THE LUNGS

BY

R. FOSTER MOORE AND H. B. STALLARD

LONDON

Case I. Mr. Foster Moore's Clinical Notes

"MRS. W., age 43 years, was sent to me on January 8, 1931, by Dr. Langford Jones with the tale that one month previously she had had a cold followed by bleeding from the back of the nose. For the last week the bleeding had ceased. For the last two weeks she had had black spots and streaks before the right eye.

She had been examined by Mr. Douglas Harmer, who could find nothing in the nose or retro-nasal space to account for the bleeding. The pupils reacted, and the trigeminal sensation was intact. There was a general congestion of the anterior ciliary vessels to the temporal side. The visual acuity was less than 6/60, and the visual field was completely missing above. A large mass in the choroid was visible with the ophthalmoscope. It extended rather widely and had a knobbly sort of surface.

In writing to Dr. Langford Jones I said I thought it likely that the mass was a metastatic growth, and asked him if he knew of any possible primary source, such as the breast for instance. He wrote at that time to say that he could find no evidence of such a primary growth. I thought, therefore, it might be tuberculous.

Three weeks later the sight became worse and the eye painful, and so I removed it. The specimen was sent to Mr. H. B. Stallard, who found a growth in the choroid which clearly was metastatic and he thought had very likely come from the lung, but as he was in some doubt, he showed it to Professor Kettle, who was of a like opinion.

It was not long before Dr. Langford Jones found clinical evidence of an intra-thoracic growth, and the patient died in June from this disease."

Pathological Report

(*a*) *Macroscopic appearances.*—Situated in the lower temporal quadrant of the choroid between the optic disc and the equator is a neoplasm, the antero-posterior diameter of which is 12·5 mm. and its thickness varies from 0·5—2·5 mm. Fig. 1 is a photograph which shows its position, shape and size.

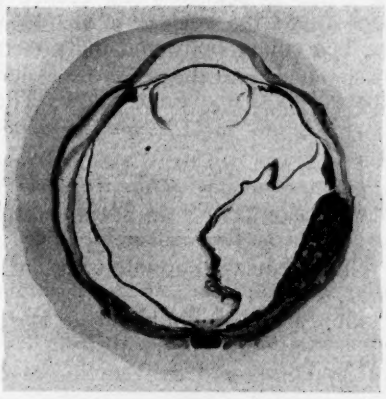

FIG. 1.

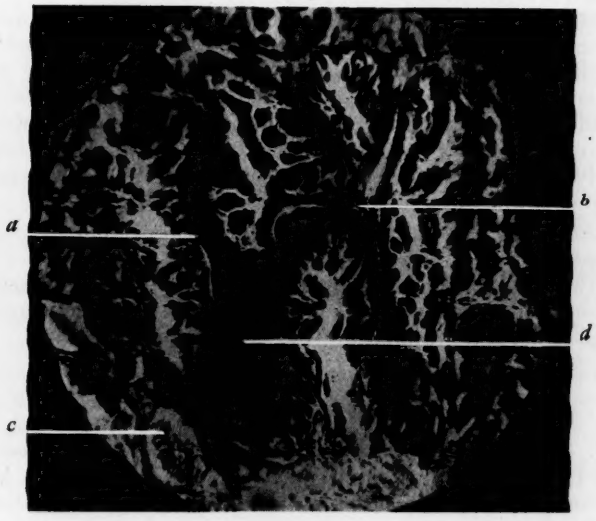

FIG. 2.

Microphotograph of part of the neoplasm in the choroid.

(*a*) *Adeno-papilliferous arrangement of* carcinoma cells.
(*b*) Connective tissue stroma and trabeculae.
(*c*) Area of cell degeneration.
(*d*) Haemorrhage.

The overlying retina is detached and rucked up. Some subretinal exudate separates the surface of the neoplasm from the detached retina. The ciliary body and the pre-equatorial part of the choroid are separated from the sclera by exudate in the suprachoroidal lymph space, both above and below. The filtration angle is open and there is no macroscopic evidence of secondary glaucoma.

(*b*) *Microscopic appearances.* The neoplasm is composed of malignant epithelial cells having an adeno-papilliferous arrangement. Some of the cells bear a resemblance to short cubical epithelial cells. They are heaped up into irregular clumps. Mitotic figures are present and there are areas of cellular degeneration. The cells have infiltrated the lymphatic spaces of the choroid.

There is a connective tissue stroma which is disposed in branching trabeculae. Capillaries and the remains of choroidal vessels are present in this stroma. The chromatophores are distorted, their processes being lost and the pigment arranged in clumps and spherules.

Bruch's membrane is stretched over the neoplasm and is degenerate. The retinal pigment epithelial cells have proliferated and some are shed into the subretinal fluid. The sclera is infiltrated at the base of the neoplasm and some carcinoma cells are present between the lamellae. There is a lymphocytic infiltration at the edge of the neoplasm. The retina over the neoplasm is detached and undergoing degenerative changes. The filtration angle is slightly narrower than normal but there is no evidence of secondary glaucoma. The optic nerve shows atrophic changes.

Pathological diagnosis.—Papillary adeno-carcinoma of the choroid, secondary metastatic deposits.

In this case it is worthy of note that the secondary metastatic deposit in the eye made itself evident before the appearance of clinical signs and symptoms of the primary neoplasm.

Case II

H. H., age 41 years, a clerk, on July 4, 1931, attended the West London Hospital on account of pain in the chest. A diagnosis of mediastinal neoplasm, carcinoma of the bronchus, was made and subsequently on July 27 he was admitted for deep X-ray therapy. On September 22, 1931, the patient noticed that his right vision was misty on rising in the morning; this became worse until he was unable to count fingers.

He was examined by Mr. H. P. Gibb who diagnosed a metastatic carcinoma of the choroid and later the eye was excised on account of pain.

The patient left hospital and was booked for another course of deep X-ray therapy late in October but he never attended and has been untraced since then.

Pathological Report

Macroscopic appearances. (See Fig. 3.) There is a neoplasm in the choroid extending from the optic disc to the filtration angle. It is flat except behind the equator where there is a large cystic space filled with blood and cell débris.

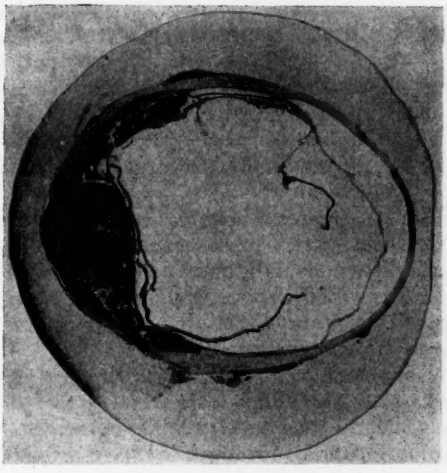

FIG. 3.

The sclera is infiltrated by the neoplasm, and the overlying retina detached and rucked up.

There is some sub-retinal exudate.

Microscopic appearances. The neoplasm is composed of columns and groups of carcinoma cells having an irregular arrangement. In some of the cell masses there is central degeneration, consisting of cell débris, pigment deposits, lymphocytes and red blood corpuscles. There is a large space immediately behind the equator measuring 9·5 × 4 mm. into which a haemorrhage has taken place. The sclera is infiltrated deeply by the neoplasm at and behind the equator.

The neoplasm extends forwards as far as the base of the ciliary body and filtration angle. The retina overlying the neoplasm is detached, rucked up and degenerate, and in the sub-retinal exudate there are some pigment cells, fibroblasts, and red blood corpuscles.

Pathological diagnosis. Metastatic carcinoma of the choroid.

A search into the literature has revealed the rarity of metastatic carcinoma of the uveal tract following a primary carcinoma in the lung or a bronchus. It is difficult to obtain accurate statistics of this disease for it is probable that some patients in the last stages of malignant cachexia die without the ocular lesion being recognized. Lagleyze states that in his experience metastatic carcinoma of the uveal tract occurs in 1 in 100,000 patients, and van der Hoeve has remarked that his predecessor Mulder had never seen a case in 35 years of practice. Since 1925 two patients suffering from this disease have presented themselves at the Moorfields Eye Hospital out of an attendance of 276,000.

Of those cases described in the literature the site of the primary neoplasm is in the breast in 65 per cent. and the lung next in 10 per cent.

Dr. Scott Pinchin (under whose care the second case, H. H., was treated) remarks that he sees each year an average of 24 patients suffering from a malignant neoplasm of the lungs, and before the case described above he had never seen a secondary metastatic deposit in the eye, the metastases occurring were frequently in the kidneys and supra-renals.

We express our thanks to Dr. Scott Pinchin and Mr. H. P. Gibb for permission to publish case II and for their help with the clinical notes, and also to Dr. R. Elsworthy (Pathologist at the West London Hospital) for his able assistance in this direction.

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METASTATIC CARCINOMA OF THE ORBIT

BY

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METASTATIC carcinoma of the orbit is an extremely rare tumour. Lagrange in his *Traité des tumeurs de l'oeil, de l'orbite et des annexes*, 1904, did not even mention metastatic carcinoma of the orbit, while Birch-Hirschfeld in one of his special chapters on *Die Krankheiten der Orbita*, 1920, published in the *Handbuch d. ges. Augenheilkunde* of Graefe-Saemisch, quotes a single personal case. Sattler in his monograph entitled *Die bösartigen Geschwülste des Auges*, 1926, although surveying the entire problem of cancer of the eye-ball, does not add anything personally to the chapter. Morax in his monograph *Le cancer de l'appareil visuel*, 1926, noted that he found published in the whole literature only twenty cases of metastatic carcinoma of the orbit and insisted upon the fact that of all cases published so far he had found but few data given concerning their histological structure. Furthermore he added to this list a personal case, unpublished, on account of its very rapid development which he neither operated on nor examined histologically. Golovine in his recent memoir entitled *Sur le diagnostic et le traitement opératoire des tumeurs de l'orbite, d'après la pratique de 40 ans*, 1930, even with his vast practical experience in this work did not describe a case that he had examined personally.

On account of these circumstances I think it is very important to have a case of metastatic carcinoma of the orbit which has been propagated from the breast published. Clinically, this case presents many different features from others that were published, chiefly in its histo-pathological appearance, and therefore it deserves to be described thoroughly.

The facts concerning the case are as follows:—

History.—C. R., aged 32 years, a housekeeper, was admitted to the ophthalmological clinic of Cluj, on May 8, 1931, suffering for about three months with a prominence of the right eyeball and a violent headache. The movement of the eyeball was very restricted in every direction and a slight diminution of sight was present.

History of the Case

The patient noticed accidentally, about two years before weaning her third child, the presence of a hard lump of the size of a walnut, in her right breast. Ten months before admission to the clinic the tumour began to increase in size and quickly involved the whole of the breast which became hard but not painful, save for occasional

shooting pains towards the shoulder up the same side. The patient noticed involvement of the axillary glands about two months later, as well as hard lumps in the supra-clavicular fossa and side of the neck, the size of which varied from that of a walnut to that of a hen's egg. Matters progressed, until about three months prior to admission, the orbit became affected. Before this the patient had consulted a surgeon who removed the breast and cleared the axilla.

On examination, the extensive scar of the breast operation was found. The supra-clavicular glands were hard and noticeable, and a hard lump was present in the middle lobe of the thyroid gland. A general medical examination revealed no further bodily signs. X-rays showed a "rugged" shadow above the right half of the diaphragm, of the size of an apple; examination of the alimentary canal showed the presence of aerophagia and aerocolia, especially marked at the splenic flexure, which allowed the left half of the diaphragm to be on the same level as that of the right half.

Rhinoscopy showed narrowing of the nasal fossa on the right side due to inflammation of the mucosa of the turbinates. The antrum of Highmore and the ethmoidal sinus were opaque to X-rays. The blood Wassermann reaction was negative.

FIG. 1.

Prior to Operation.

Ophthalmological Examination.—The left eye was normal. The anterior parts of the right globe were normal save for a convergent squint of 30° with sursumvergence added. There was no exophthalmos. (Fig. 1.)

Palpation of the lower margin of the right orbit disclosed the presence of a hard elipsoid tumour adherent to the orbital floor. This tumour measured about 2 cm. in length and 1 cm. in breadth, it was nodular and painless and it extended into the depth of the orbit. This tumour had no connexion with the eye-ball which it

FIG. 2.

After Removal of the Growth.

had displaced inwards and slightly forwards. Movements were much restricted, especially in an outward direction. The field excursion was: 45° upwards, 40° inwards, 45° downward and 0° outwards. Ophthalmoscopic examination demonstrated the presence of a slight oedema of the papilla. Visual acuity was R.E., 5/10, L.E., 5/5. The patient made no complaint of diplopia. The refraction was emmetropic and the visual field was normal.

On May 15, 1931, the tumour was extirpated by a transpalpebral inferior orbitotomy. At operation the tumour was found to be closely related to the periosteum. The tumour was encapsulated and reached upwards under the inferior rectus muscle to a distance of approximately 2 cm.; in the depth of the orbit was a large vascular pedicle. The operation was completed by primary suture.

Four days later the wound was healed and the stitches were removed. A fortnight after admission the patient left the hospital, with her eye in an approximately normal position (Fig. 2); convergent

strabismus of 10° remained, but movements in an outward direction had improved to 30°. The acuity of vision had improved to 5/7. The ophthalmoscopic appearances were unchanged.

Anatomico-pathological Appearances.—The tumour was of the size of an almond with the major axis antero-posteriorly; it was encapsulated and a fibro-vascular pedicle was present at the posterior end. Thin radiating septa reached from this pedicle into the tumour mass dividing it into lobules. These lobules exhibited three different histological appearances:—bulky lobules, lobules with alveolar arrangement and others with a glandular arrangement. The bulky lobules were most numerous and occupied the posterior pole of the tumour. The stroma appeared as a thin fibrous structure, while the cellular parts were typical of a basal celled *carcinoma*, containing many polygonal cells. (Fig. 3). The alveolar and glandular lobules were localized particularly at the periphery and in the anterior part of the tumour. At this level the stroma was better developed forming large compartments which appear in transverse and longitudinal section. The neoplastic elements in places appear as cellular islands localized in the middle of alveolar tubules, while in other places can be seen cellular bands, which occasionally present the appearance of a gland with central lumen.

At the periphery of the tumour in the capsule can be distinctly seen evidence of neoplastic invasion of the lymphatics and of the orbital fat, under the guise of numerous epithelial islands with an eccentric dissemination (Fig. 4). In no part of the growth was there any evidence of degeneration, inflammation, haemorrhage or other vascular alteration.

Summarising the above case it may be stated that a woman aged 32 years, had a carcinoma of the right breast two years ago, which later on became extensively spread through the entire breast and produced metastases in the axillary lymph nodes, thyroid and lung, and later a metastasis of the cellulo-adipose tissue around the floor of the right orbit and probably also the ethmoidal sinuses and right maxillary antrum as well. At this time, the right breast was extirpated including the corresponding axillary lymph nodes. Three months later, after the breast was extirpated, the metastatic tumour appeared upon the floor of the orbit associated with a convergence and sursumvergence of the right eye, with a limited motility on the temporal side, slight papillary oedema and a diminution of visual acuity. Although the operation showed that the orbital metastasis preserved a close contact with the floor of the orbital periosteum, it affected neither the periosteum nor other parts of this region but maintained itself in the encapsulated cellulo-adipose tissue of the orbital cone under the right inferior rectus muscle.

After the orbital tumour was extirpated, the eyeball began to

FIG. 3.

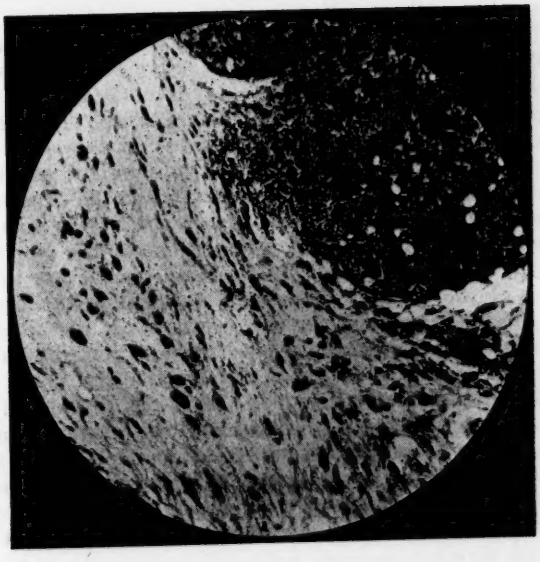

FIG. 4.

regain approximately its normal position maintaining a convergence of 10°, but it recovered a motility of 30° on the temporal side, and a great improvement in visual acuity.

The histological examination of the extirpated tumour showed an encapsulated baso-cellular carcinoma, with a diffuse massive infiltration on the posterior side, and with a trabecular or even glandular arrangement of cells anteriorly. The neoplasm eccentrically had the appearance of a cancerous lymphangitis of the orbit.

* * * *

This case of metastatic carcinoma began, as in the majority of cases after a breast cancer, a year and nine months after the primary growth was detected. It appeared at an early age (at 32 years old) compared with other cases of which the majority were 40 years and even older. During the progress of this case we noticed two interesting clinical facts, namely, the existence of a slight oedema around the papilla on the affected side associated with diminution of sight, which after the tumour was removed returned to normal, and the appearance of the convergence associated with great diminution of motility on the affected temporal side, which became ameliorated after the operation.

The appearance of the papillary oedema and the diminution of sight during metastatic carcinoma of the orbit was observed also by some other authors. For instance, Axenfeld (1907) noted in one of his cases that the papillary stasis was due to a tumour which filled the entire orbital cavity, compressing the optic nerve. In Birch-Hirschfeld's case (1915) the metastasis in the orbit developed in the cone formed by the muscles, compressing early the optic nerve at its anterior segment, without invading its dural sheath and producing an atrophy. In Finoff's case (1921) the papillary oedema with venous stasis, and the diminution of sight which appeared at the same time with an exophthalmos and terminated in blindness, gave the impression of the existence of a tumour of the optic nerve. In Morax-Guégo's case (1922) the metastasis of the orbit was associated with a complete loss of sight of the affected eye through the invasion of the optic nerve, whose papilla on ophthalmoscopical examination showed venous dilatation and a reduced calibre of the retinal arteries. In Magnus's case (1929) the metastasis occupied the apices of both orbits, producing a bilateral papillary stasis, and histological examination showed that the neoplasm had penetrated the meningeal sheath of the optic nerve through the tracts of some small vessels, without producing any degeneration of the nervous elements. In Kreibig's case (1931) the cancerous metastasis of both orbits provoked a progressive papillary oedema, followed by a post-neuritic atrophy

of both optic nerves and by blindness due to the optic nerves being compressed in the apices of the orbits without the nerves or their dural sheaths being penetrated by the growth. These cases show, therefore, that almost all the papillary alterations are associated with some visual disturbance (except Morax and Guégo's cases, neither of which were verified by operation or histological examination, and the case of Magnus in which histological examination proved the invasion of the optic nerve sheath); due not to the invasion of the optic nerves and their sheaths by the new growth, but to pressure strangulation from a well developed metastasis in the interior of the muscle cone, especially close to the apex of the orbit.

In my case, though the metastasis in the orbit did not have a large dimension and was situated more on the anterior side of the orbital floor outside the cone of muscles it caused disturbances in the circulation of the optic nerve, and produced diminution of visual acuity. The explanation of this circulatory disturbance of the optic nerve caused by a tumour of small dimensions which did not produce any marked exophthalmos, is that the metastasis was localized at the anterior part of the orbital floor, and exactly in the vicinity, or very close to, the penetrating point of the central vessels of the retina into the interior of the optic nerve where the tumour must have led to compression of these vessels. The mechanical effects of a tumour at this site also induced a strabismus.

Evidently this explanation is supported by the rapid improvement in visual acuity which occurred in the affected eye after the tumour was extirpated. The case showed that a metastatic cancer on the floor of the orbit must be removed as early as possible. In this way visual disturbance can be avoided, as was noticed in our case, or blindness will ensue.

The convergence and sursumvergence strabismus associated with loss of the greater part of the eyeball movement on the temporal side, disappeared to a large extent after the tumour was extirpated and is of interest. Evidently the paralytic symptoms of the ocular musculature which appeared during the progress of this orbital metastatic carcinoma, made Elschnig's case a good subject to study, who asked himself if these disturbances were of neural origin (by compressing the oculo-motor nerves before their branches penetrated the muscles) or of muscular origin (brought about by pressure of the tumour on the muscles). The majority of authors who have had occasion to study such cases conclude that the paralytic ocular symptoms are in reality almost always of muscular origin due to neoplastic muscular invasion and in consequence a simple atrophy. The histological researches by Horner (1864), Elschnig (1898), Wintersteiner (1899), Axenfeld (1907), and Magnus (1929) (who have found cancerous invasion in various degrees in all the ocular

muscles, their perimysium and their blood vessels), established the fact that the ocular musculature constitutes the site of election for localization of carcinomatous metastatic deposits in the orbit. Wintersteiner in the same way affirms that focal centres of cancerous metastatasis in the ocular musculature are more frequent than is believed, especially in the cases with multiple metastases, and that often the clinical disturbances remain in a latent state on account of the small focal centres. Finally Sattler, in one of his synthetic studies in connection with carcinoma of the eyeball, concluded that the localization of the metastasis in the ocular musculature constituted a general rule and that a direct orbital localization was exceptionally rare.

From all these researches there is another conclusion, *i.e.*, that cancerous metastatic invasion of the orbit takes place through the vascular circulation. If we attempt to explain the ocular appearance of static and motility disturbances of our case, and the amelioration of the greater part of the symptoms after the extirpation of the tumour, we see the difficulty in interpreting our case because the pathogenic conceptions given above do not permit us to explain the past features and functional operation of these disturbances.

For the interpretation of this case it must be assumed that the important rôle in provoking the ocular disturbances must have been played by the mechanical obstacle which the tumour produced upon the contraction of the ocular musculature, probably by the cancerous lymphangitis, proved at the histological examination, and not by the cancerous infiltration of the musculature as is usually believed. This interpretation cannot be wrong, because our case is not the only one. Evidently Kreibig in a case recently described, also noticed the appearance of the bilateral disturbances of ocular motility, and the histological examination of the entire orbital contents, which were eviscerated, did not prove any focal invasion of the musculature by growth, but only the existence of an orbital cancerous lymphangitis. My case, together with Kreibig's recent case, are among those extremely rare cases in which the metastatic cancerous invasion occurred through the lymphatic tracts, and involved not only the ocular musculature, but also the orbital cellulo-adipose tissue, and especially the lymphatic tract, where the histological appearance was of a cancerous lymphangitis. The metastatic tumour which was extirpated in this case, pertained to the cellulo-adipose tissue around the orbital floor. The histological study of this tumour showed some interesting peculiarities. However, this histological examination showed also that the tumour had a dominant structure of an atypical form of breast cancer, because it was composed of diffuse epithelial masses, or of bands among which the glandular forms constituted the isolated

remains localized towards the periphery of the tumour at the anterior pole. This histological structure indicates to us that the posterior pole of this tumour with its massive atypical cancer, was in reality the oldest part of the tumour, and from there growth took place and propagated till it reached the inferior margin of the orbit, where it resembled epithelial bands with a glandular appearance, characteristic of the earlier phase of this kind of tumour. The fact that in the whole extirpated tumour mass we have not noticed any alterations of blood vessels, but an extensive cancerous lymphangitis, showed that the orbital metastasis of our case was through the lymphatic tract.

One histological point that differentiated our case from others which have been published, was the appearance of a very cellular tumour with its stroma reduced to a minimum, while all other authors insist upon the scirrhous character of the cancerous metastasis of the orbit, especially when the place of origin was in the breast. This encephaloid appearance characterizes especially those cancers with a very rapid progress, as occurred in this young woman, and corresponds with the whole clinical evolution of the case.

The complete lack of inflammatory reaction of the stroma in the orbital cellulo-adipose tissue, and the absence of necrotic centres and haemorrhages in the tumour mass indicate the recent appearance of the entire orbital metastasis and emphasize in a more detailed manner its histological physiognomy.

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AN UNUSUAL RESULT FOLLOWING TRAUMATIC IRIDO-CYCLITIS

BY

D. J. WOOD

CAPE TOWN

THIS patient came to me with irido-cyclitis in each eye following operations for cataract. After six months treatment her left eye was operated on—discission—and fair vision was obtained. The right had not improved however.

There was much general redness, but the cornea was fairly bright, so that one could easily see new vessels all over the iris. There was a small coloboma above, but vision was very poor, and there did not seem to be any justification for attempting any operative interference. When I suggested excision the patient agreed at once, glad to be rid of a continually weak, watery, and intermittently painful eye.

Though I had examined her several times with the slit-lamp, I did not think of estimating her corneal thickness, and the interesting pathological condition, found afterwards, was not anticipated.

Over the whole of the back of the cornea was a thick layer of new tissue, thinnest at the edge, but in the centre reaching a thickness two-thirds of that of the cornea.

The deepest parts next to Descemet's membrane are well formed layers of fibrous tissue, the more superficial layers are more granular in texture, contain fewer nuclei, but more particles of uveal pigment, and many blood vessels. There was no layer of endothelium, and when the eye was excised one could see no precipitates, though when I first saw her there were plenty.

The interesting feature is the great transparency of the tissue which did not reveal its existence to myself, or any of my colleagues who saw her. None of us thought of looking at the thickness of the light track through the cornea, otherwise one must have noticed the abnormality.

I have seen a similar condition in the angle of the anterior chamber in a case of leprosy, but there corneal opacity did not permit any vision into the eye, and one could not say whether the new tissue was clear or not.

Now if nature can, under certain conditions, produce clear fibrous tissue, is there not a chance that by imitating these conditions we might get clear scars on the cornea instead of opaque.

One condition would seem to be that the tissue is laid down in the aqueous and friction is absent.

Acting on this idea, I obtained from the depot of Messrs. Zeiss in Johannesburg, a set of their contact glasses with the idea of

FIG. 1.

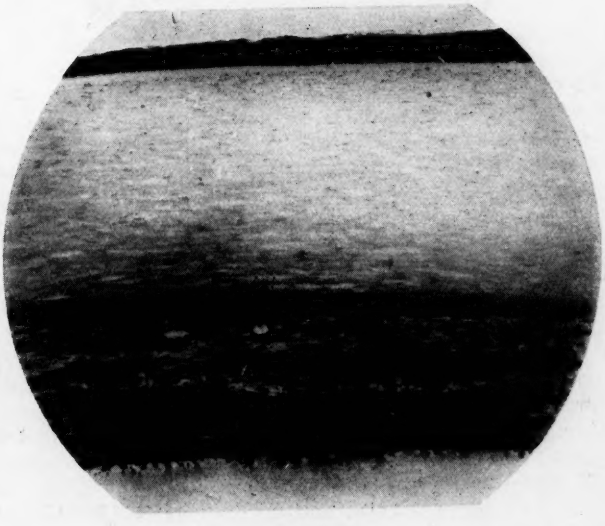

FIG. 2.

trying them in cases of corneal ulcer. I was, however, unlucky in having only one case during the time I had the contact glasses, and she was unmanageable, and continually got the contact glass out of place. I would like to suggest that some of my colleagues better situated might try the experiment again. After all, one should be in a favourable position to get a cure of an ulcer if one could protect it, and keep it bathed in a sterile natural fluid.

The photographs show (1) the angle of the anterior chamber with a finely granular coagulum, doubtless the source of the

FIG. 3.

fibrinous deposit. At the circumference of the cornea above is the usual mass of lymphocytes found in all cases of chronic iridocyclitis.

(2) The centre of the cornea showing the thick formation of fibrous tissue on the membrane of Descemet. It contains cells, extra-cellular pigment, and blood vessels.

(3) Deposit behind Descemet's membrane in a case of leprosy. The convoluted line of the membrane is visible. The tissue on it is fibrous tissue with many cells. A single precipitate is shown.

Although there were lymphocyte cuffs round the retinal vessels and plenty of well formed plasma cells in the anterior part of the choroid there were no lymphocyte nodules in it, no marked destruction of pigment cells, and no giant cells. In short, it was not a case of sympathetic ophthalmitis.

A CASE OF FILARIASIS OCULI

BY

K. KOMAN NAYAR, D.O.M.S.

ACTING SUPERINTENDENT, GOVERNMENT OPHTHALMIC HOSPITAL, MADRAS

AND

A. KANDASWAMI PILLAI

IN CHARGE, EYE DEPARTMENT, GOVERNMENT HEADQUARTER HOSPITAL, CALICUT

KUNHI AHMED, police constable, aged 34 years, reported himself at the Eye Department of the Government Headquarter Hospital at Calicut, Malabar district on June 10, 1931, with the complaint that his left eye vision was blurred and that he had noticed a worm-like object floating about in front of his left eye for the past five days. He did not complain of any symptoms other than a feeling of heaviness on the left side of his head. His medical history sheet showed only three previous admissions into the hospital for malaria and three for bronchitis. His general health was good.

He was examined by Dr. A. Kandaswami Pillai with the following result:—

R.E.V.=6/6; L.E.V.=6/18, not improved with glasses.

Both pupils active to light and accommodation; tension normal in each eye. The pupils were subsequently dilated with homatropine and adrenalin for fundus examination.

Right eye: Fundus normal; media clear.

Left eye: In the vitreous, just in front of the macula, was seen a thread-like, wriggling worm, coiling itself and moving briskly, especially when light was thrown directly on it. There was an oval clot of blood, about three-fourths of the size of the optic disc, near the macula, to which one end of the worm was attached; the other end of the worm kept on moving in different directions in the posterior part of the vitreous. The worm appeared to be, roughly about 3 mm. long at that time. At times a sheath could be made out, as also what appeared to be a few silvery white bands across the worm. Whether these bands were due to reflection of light from the convex surfaces of the loops formed by the moving worm, it is difficult to say.

Blood showed a haemoglobin percentage of 90, and the result of a differential count was:—

	Per cent.
Polymorphonuclear	56
Lymphocytes	26
Large mononuclear	4
Eosinophiles	14 (not at all a high percentage among the general population of that district)

Night blood—negative for microfilariae.

Physical examination: No enlargement of glands anywhere in the body; no other signs of infection with filaria.

At the request of one of the authors (K. K. N.) who was shown the case while at Calicut on a holiday and verified the ophthalmoscopic findings, the patient was sent over to the Government Ophthalmic Hospital, Madras for further observation and investigation.

On June 20 and subsequently, before sending the patient to Madras, Dr. A. Kandaswami Pillai noted that the movements of the worm were getting slower, the patch of retinal haemorrhage was getting smaller in size and the worm was gradually getting longer.

The patient reached Madras on the morning of June 24, 1931, after a railway journey of over 400 miles. On arrival he gave the history that sometime during the previous night, when awake, he suddenly saw flashes of red and green sparks in front of his eye and that afterwards, he lost sight of the moving object in front of the eye.

On external examination, both eyes were found normal in appearance.

R.E.V.=6/6, partly; L.E.V.=6/6, partly, not improved with glasses.

Pupils active, right fundus normal and media clear.

In the left fundus, the remains of the old haemorrhage were seen near the macula. There were also numerous shiny white spots in the vitreous just in front of the macular region, but no sign of the worm could be made out in the eye.

Examination of the peripheral blood of the patient taken at night on different occasions showed microfilaria (bancrofti).

The patient was under observation daily and the haemorrhage was found to get smaller in size and the vitreous in front of the macular region clearer.

On June 29, the worm was found lying in the bottom of the anterior chamber and moving about briskly when light was thrown on it. It was greyish white in colour and was approximately 2·5 cm. in length and less than 0·5 mm. in width. The appearance was that of an adult filaria. Details could not be made out by biomicroscopy as the worm became very active when light was thrown on it.

The patient was kept under observation for a few days and the worm was daily seen lying at the bottom of the anterior chamber. The mobility of the worm remained much the same from day to day. The patient observed the presence of the floating object in front of his eye now and then.

On July 2, it was decided to remove the worm from the anterior

chamber; after the usual preliminaries, a small keratome incision was made at the limbus below; the aqueous rushed out through the wound, evidently carrying the worm with it, but unfortunately the worm was lost in spite of all precautions. The anterior chamber was refilled with normal saline but no trace of the worm could be made out. On July 10, the patient was discharged from the hospital with the eye quiet, with a vision of 6/9, improving to 6/5 save one letter with +0·75 D. sph. (tested while the pupil was still dilated). The anterior part of the eye, as examined with the corneal microscope, was found to be normal.

The patient came from a district where filariasis is fairly common; his peripheral blood contained microfilaria bancrofti. The size of the worm and its appearance leave little doubt that it was an adult filaria. Although the measurements of the worm as judged from its appearance in the anterior chamber are approximate, they are within the limits of the measurements given by Cruickshank and Wright in their investigation "Filariasis in Cochin" (Malabar coast) in 1914. (Ref: *The Indian Journal of Medical Research*, Vol. I, No. 4, 1914.)

The case is interesting on account of its rarity—only a few cases having been recorded so far in the human eye, of which a list is given by Elliot in his book "Tropical Ophthalmology." Unfortunately, in this case, just as in those quoted, the nature of the parasite was not determined with certainty. Probably no case has been recorded till now in which a filaria was seen emerging from the retina or subretinal space gradually pushing itself forward into the vitreous and then disappearing altogether for a few days before making its appearance in the anterior chamber, where it remained free and alive for some days without causing any reaction in the eye or any inconvenience to the patient.

I discussed the case with Lt.-Col. R. E. Wright, I.M.S., on his return from leave and submitted my records and a drawing. He added the following note, which I quote in full:—

"As far as I know there has not been any case of an intra-ocular filarial worm reported which has a greater claim to be regarded as bancrofti. The patient came from an area where bancrofti and only bancrofti is endemic, there were microfilariae bancrofti in the blood. The thickness, length and conformity as far as I can gather from your numerous observations, place it within the limits of the series of adult filariae described and figured by Cruickshank and me in 1914, the length of smallest worm being 20 mm. These were taken from lymphatic glands of patients in an adjoining district; we did not see any intra-ocular filariasis during our survey. R. E. Wright."

PARALYSIS OF OCULAR MUSCLES FOLLOWING SPINAL ANAESTHESIA

BY

MAJOR J. BIGGAM, R.A.M.C.

BANGALORE, INDIA

AT a large military hospital in England, between January and July, 1927, I examined five cases of paralysis of one or other of the recti muscles of the eye following on the use of stovaine as a spinal anaesthetic.

Although stovaine has been extensively used as a spinal anaesthetic in this hospital since 1915 there is no record of any case of paralysis of ocular muscles following its use, with the exception of these five cases which occurred early in 1927.

Until this date the stovaine used was obtained from one firm, and, after the occurrence of the cases, analysis of the supply by this firm revealed nothing deleterious.

Since July, 1927, the stovaine has been obtained from another firm and up to July, 1931, no further ocular complications have arisen.

No important alteration in the method of administration or in the dosage employed has been made during the 16 years under review.

The following are short notes of the individual cases:—

Corporal G.S., aged 22 years, Disease—Chronic Appendicitis

January 29, 1927. *Operation—Appendicectomy.*
January 31, 1927. Vomiting and jaundice.
February 3, 1927. Headache. (There is no note of when this started.)
February 10, 1927. Complaints of diplopia only on looking to the left.
February 14, 1927. I saw the patient for the first time. He had a slight but definite paresis of the left external rectus muscle. He had single vision when looking straight ahead. The pupils were equal in size and normal in reactions.
February 20, 1927. On re-examining the patient I found no trace of muscle paresis nor could diplopia be elicited by any test. The Maddox rod showed no distant muscle balance error. Complete examination showed no eye abnormality except one dioptre of myopic astigmatism in each eye.

I lost touch with this case from February 20, 1927 until May 31, 1927, when I heard from a doctor in Leeds that "He is attending me now for very intractable headache and he still has some weakness of the eye muscles with diplopia if he tries to look to the extreme left." As far as I know he had not been examined again by an oculist. Unfortunately I heard no more of this case.

Driver H.M., aged 21 years, Disease—Subluxation Internal Semilunar Cartilage

March 5, 1927. Operation—Excision of internal semilunar cartilage 10.30 a.m.
March 6, 1927. Intense headache, frontal, occipital, and down the back of the neck started at 2 a.m.
March 11, 1927. Headache continued. At 3 p.m. he woke from sleep with double vision. Examination showed a marked paralysis of the left external rectus. He had single vision only on looking to the extreme right.

March 15, 1927. He still had slight continuous frontal headache. The diplopia was unchanged.

March 23, 1927. Diplopia only on movements of the eyeballs to the left of the middle line.

March 28, 1927. Diplopia only on movements of the eyeballs to the extreme left.

March 30, 1927. Double vision completely gone. Complete examination revealed no other abnormality. Patient stated that his headache continued, although gradually diminishing in intensity, till March 27, 1927, and then almost abruptly disappeared.

July 4, 1927. The patient, who had been discharged from the Army in the meanwhile, was traced into a non-medical government institution the medical officer of which reported "no return of double vision since his reception here on June 3, 1927."

Sergeant S.A.S., aged 30 years, Disease—Chronic Appendicitis

April 12, 1927. Operation Appendicectomy—Anaesthetic stovaine, supplemented by a partial general anaesthetic.

April 14, 1927. Headache started. He also complained of stiffness in the back of the neck and sleeplessness.

April 15, 1927. He first noticed the diplopia. Examination revealed a marked degree of paralysis of the left external rectus. Except for the paralysis of this muscle no other eye abnormality external or internal was found. He still complained of "dragging pains" in the back of the neck.

April 21, 1927. Diplopia unaltered. Headache still persisted.

April 25, 1927. Headache had disappeared. Muscle paralysis improving slightly.

April 28, 1927. Diplopia only on looking to the left of the middle line.

May 5, 1927. He felt fit, but there was no change in the muscle condition since April 28, 1927.

May 15, 1927. Condition unchanged. Complete examination revealed no defect other than that of the left external rectus muscle. He was discharged from Hospital and given leave of absence.

May 23, 1927. According to a letter from the patient, the diplopia is improving.

June 24, 1927. He stated in his letter that the diplopia has now entirely disappeared, the end point of the disappearance of the double vision coming rather suddenly and unexpectedly.

Driver G.W., aged 22 years, Disease—Acute Appendicitis

May 11, 1927. Operation—Appendicectomy.

May 13, 1927. Some frontal headache since operation but no eye trouble until this morning when he noticed diplopia. Examination revealed slight but definite paresis right external rectus muscle.

May 21, 1927. From the 13th until this date severe headache was a marked feature of the case. The muscle paresis and diplopia remained unaltered.

May 31, 1927. The headache gradually lessened in intensity and the muscle paresis improved.

June 4, 1927. He felt fit. Headache had disappeared. He did not now complain of diplopia, which however could still be readily elicited with red and green glasses when he looked to the right.

June 10, 1927. He had no symptoms. Doubtful diplopia could be elicited with special tests on extreme eye movements to the right. He was discharged Hospital for leave.

Corporal A.R.C., aged 28 years, Disease—Subacute Appendicitis

July 1, 1927. Operation—Appendicectomy.

July 2, 1927. Severe headache which continued for a week gradually diminishing in intensity.

July 9, 1927. He noticed diplopia on wakening. Examination revealed marked left external rectus paralysis. He had single vision only when looking to the extreme right. Little change was noticed for 3 weeks and then gradual improvement began.

August 9, 1927. Could now see single when looking straight ahead. He still had diplopia on movements of the eyeballs to the left of the middle line.

August 18, 1927. He did not complain of diplopia which however could easily be elicited with special tests when he looked to the left.

August 28, 1927. Very slight diplopia still remained.

September 18, 1927. Complete disappearance of diplopia. Nothing abnormal noticed on complete examination of each eye. The patient had no headache and felt fit.

CASE.	G.S.*	H.M.	S.A.S.	G.W.	A.R.C.
Number of days elapsing between operation and onset of paralysis - - -	12	6	3	2	8
Number of days elapsing between onset and disappearance of paralysis - -	10	19	70	28	71
Eye affected - - - -	L	L	L	R	L

* NOTE.—The figures given for the case of G.S. are probably unreliable. He was not under my observation at the time of the onset of the muscle paresis, and he was lost sight of six days later. The other cases were under my observation from the beginning, and were observed, or traced, until the condition had completely cleared up.

Summary

1. The occurrence, under the conditions mentioned, of an isolated group of five cases within six months suggests some toxic element in the drug in use during this period.

2. All cases complained of headache, usually very severe, which preceded the onset of muscle paralysis by one to seven days. The headache lasted from one to three weeks.

3. The paralyses were maximal at their onset, remained stationary for varying periods, and then gradually improved. In the case of G.S. complete recovery appears to have been followed by relapse, about which, however, no accurate information is available.

4. In one case the paralysis occurred not more than two days after the administration of the drug.

5. In no case was any abnormality, other than that of the muscle affected, discovered.

ACUTE GLAUCOMA: AN HISTORICAL NOTE

BY

ARNOLD SORSBY

LONDON

WHILST the term glaucoma goes back to the Hippocratic writings, the conception of acute glaucoma is barely more than a 100 years old. It is true that descriptions suggestive of the affection are found in early writings. Thus Hirschberg[1] draws attention to passages in Tabari, and Benevenutus and particularly to the significant passage in Sams-addin:

"Migraine of the eye, also known as Headache of the pupil. This is a pain situated deep in the eye stabbing or pressing or both; sometimes constant, sometimes transient. It is caused by retained matter in the vessels of the retina. The aforementioned pain is its sign. Hemicrania precedes it or is associated with it. Dullness of the fluids is always present. Sometimes cataract develops from it and also dilatation of the pupil. If it becomes chronic, tenseness of the eye follows and vision is feeble. Treatment: bleeding and purgation. Instilling white of egg and milk; sometimes drugging for the pain is necessary."

Sams-addin's attempt at differential diagnosis within the group of lesions passing under the name of ophthalmia had no far-reaching consequences, and glaucoma itself remained a vague conception for irremediable blindness in which the pupil had a bluish or greenish discolouration. Indeed glaucoma was essentially hopelessly incurable blindness for in the medical philosophy of Galen which held sway till the Renaissance, glaucoma was dryness and shrinking of the crystalline (lens), itself the seat of vision. The reorientation that resulted, in the first place from the work of the XVIIth century anatomists and physiologists, especially of Keppler and Schreiner in showing the retina as the organ of sight, and secondly from the conclusive evidence of Brisseau in 1709 that it is the lens that is affected in cataract and not a space in front of it, demanded a new conception for the pathology of glaucoma. Never a very definite clinical entity, it passed through a period of exceptional vagueness during the whole of the XVIIIth century and it is only towards the beginning of the XIXth century that glaucoma commences to re-assert itself. It is significant that in its re-emergence it is a clinical entity which is first recognized—under a multitude of different names—and only later is it recognized as glaucoma. The essential unity of acute and chronic glaucoma comes later still, and only after the unfortunate name had lost all its original significance and had become a mere label.

During the XVIIIth century and for nearly half of the XIXth century glaucoma remained a descriptive term for that phase of ocular inflammation in which the pupil assumes a greenish hue. Tentative attempts at the recognition of what we know now as acute glaucoma were made as early as 1722, when St. Yves[2] described an acute condition in which blindness with an unobscured pupil supervenes followed afterwards by glaucoma.

"La goutte sereine suit cette fluxion après laquelle succède un glaucome."

The anonymous author (?John Hill) of the *Fabrick of the eye and the several disorders which obstruct the sight.* London, 1758,[3] gave an account in which glaucoma is divided into three stages, of which the first is an unconvincing description, and the second and third stages indicative of absolute glaucoma with its acute symptoms.

Under the name of glaucoma Arachart[4] in 1786 describes acute conditions in which there is varying degree of greenish hue; perfect glaucoma is present when there is complete blindness and the pupil is greenish-yellow, approaching the colour of a dead oak-leaf; imperfect—when there is still some vision and the pupil has a whitish—greenish colour.

It is not until 1813 that a really convincing description of acute glaucoma appears—an account by Joseph Beer.[5] A form of iritis is differentiated from other varieties by its distinctive symptoms and in that it ends in blindness, a greenish hue (glaucoma), a dilated pupil and cataract—a passable description of absolute glaucoma. In his ambitious attempt to describe eye diseases on a basis of causation he named this acute eye condition iritis of gouty origin.

A most excellent description is given in 1818 by A. P. Demours.[6] Rainbow colours and hardness of the eye are mentioned and the condition is named glaucoma. An even fuller account is given by Weller[7] who combines the descriptions of both Beer and Demours. He retains both terms—arthritic iritis (and ophthalmitis) and glaucoma, apparently as separate clinical entities, discussed in different parts of the book; the same condition seems to have been regarded as arthritic ophthalmitis when there was no greenish pupil reflex and glaucoma when such was present.

In this connection it is worth noting that whilst a strong resemblance between acute iritis and acute glaucoma was tacitly admitted in the name of arthritic iritis, the various synonyms suggested such as arthritic ophthalmitis, (Benedict, Rosas) arthritic inflammation of the internal tunics (Lawrence, Wharton Jones), show that a realization of some fundamental difference between the two conditions was gradually being achieved. How difficult that must have been, before the full appreciation of the significance of hyper-

for this variety of iritis being much less amenable to antiphlogistic treatment than the others.

2. *Changes in the iris and pupil.* Beer has described these as varying in two different habits of body. In those who are of a meagre and irritable habit, and tense fibre, the pupil contracts, is filled with effused lymph, and becomes adherent to the capsule, as is generally the case in the other species of iritis. In such cases, the only characteristic symptom, besides the white ring round the cornea, is a varicose state of the blood vessels of the iris, so that after the disease has fully developed itself, they may be discerned ramifying on the surface of that membrane, or forming a vascular wreath within the verge of the contracted pupil. Before it arrives at this stage, the inflammation is always attended with general fever. If the eye is left to itself, it does not suppurate, but its contents begin to be absorbed, and at last its volume is extremely diminished. In those, ~~again~~, who are of a gross habit of body, possess little sensibility, and have a lax fibre, the iris instead of expanding, contracts remarkably, a sign of attending amaurosis, and at the same time loses its motion and natural black colour. The pupil is not always dilated uniformly along its whole circumference; not unfrequently the iris contracts more towards the temporal and nasal sides of the eye, so that the pupil assumes an oval shape; indeed, the iris sometimes becomes so narrow on the two sides mentioned, especially on the temporal, as almost to disappear. Along with these changes, there is no effusion of lymph, nor any abscess on the surface of the iris. Behind the enlarged pupil, there is perceived the greyish-green reflection, characteristic of glaucoma, a state of the eye depending on absorption of the pigmentum nigrum, with dissolution of the vitreous humour, and occasionally accompanied by discolouration of the lens. After a time, the lens is plainly seen to have lost its transparency, and to have assumed an opaque sea-green colour; it swells considerably, and projects through the pupil, into the anterior chamber. The iris, lying upon the enlarged lens, seems much altered from its natural texture; it looks soft, and as if it had undergone a degree of maceration. The varicose state of the vessels of the conjunctiva increases, while those of the choroid becoming similarly affected, form bluish knots, which shine through the sclerotica. The anterior part of this tunic being extenuated by the pressure of the morbid parts within, a dark ring shines through it, exactly occupying the

PAGE from the first edition of William Mackenzie's *Practical Treatise of the Diseases of the Eye*, London, 1830, with his marginal notes for additions to the second edition.

In speaking of arthritic iritis he states: "The general symptoms of iritis are present in the arthritic species, namely, zonular sclerotitis, discolouration of the iris, turbidness of the pupil. with changes in its shape, size and mobility, impaired vision and pain in and around the eye. These symptoms are, however, modified in such a manner as to afford a ready diagnosis."

This reproduced page with its reference to Acute Glaucoma of Lawrence deals, as can be seen, with the diagnostic features in the changes in the iris and pupil. "Middleton's case, March, 1883," probably refers to a patient of that name.

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tension, can be imagined from the difficulty in diagnosis that cases of subacute glaucoma may present to-day.

The first to realize the connection between arthritic ophthalmitis and glaucoma seems to have been Sir William Lawrence. His work on glaucoma has received no consideration from historians, yet it is of sufficient merit to add lustre even to his name. In the lectures delivered at the London Ophthalmic Infirmary and published in the *Lancet* for 1826, he, like Weller, describes arthritic ophthalmitis and glaucoma as separate conditions, giving under the one a good account of acute glaucoma, mentioning hardness of eye as one of the symptoms, and under the other an account of chronic glaucoma, in which the cardinal sign, increased hardness, is missing.

In speaking of deep seated acute inflammation of the eye he follows a current conception in distinguishing ophthalmitis arthritica from idiopathic ophthalmitis. Ophthalmitis arthritica "ends in a dilated pupil, that is the appearance called glaucoma; it might be called in contradistinction to the slowly occurring change of similar character which will be afterwards described, acute glaucoma. Benedict has named it, in consequence of its frequent occurrence in gouty persons, ophthalmitis arthritica."[8] In discussing glaucoma (*i.e.*, blindness with a greenish pupil) he again brings out its association with arthritic ophthalmitis.

"With respect to the causes of this affection, we can point out nothing peculiar as contributing to its occurrence. It takes place at or after the middle period of life, and in persons not of the most healthy character. It appears to me to be merely a chronic form of the same inflammation which I have described to you as the arthritic inflammation affecting the posterior coats of the eye; the changes which occur in the retina, vitreous humour and lens, as the consequences of that inflammation, are rapid and sudden, whilst in the present case the disease has a slower progress, and the changes are more slowly produced; but it certainly does appear to occur more frequently in such persons as are liable to gout and rheumatism than in others. It is of considerable importance that you should be well aware of the characteristics of this affection, since it is liable to be mistaken for cataract."[9]

In lectures delivered at St. Bartholomew's Hospital, eye diseases are dealt with less fully. As reported in the *Lancet* for 1830, in speaking of acute inflammation of the vitreous humour, retina and choroid (*i.e.*, the "internal tunics" of the earlier lecture) he stated: "These occur in elderly persons, at all events in persons past the middle period of life; severe pains will come in the eye, the pupil becomes dilated and instead of presenting its natural colour, exhibits a dull dirty greenish hue, in consequence of the change which the inflamed vitreous humour has undergone. With

this green shade, this excessively dilated condition of the pupil and the dull leaden looking iris, you have almost a complete loss of vision. It is in fact, an inflammation commencing in parts most essential to the function of vision in the retina, in the vitreous humour and probably involving the choroid coat. It has sometimes been called Acute Glaucoma, this term being derived from the greenish appearance of the eye. It has been called glaucoma from another symptom which takes place where, without any enlargements of the vessels, without any very severe pain or absolute extinction of vision in the first place, the pupil exhibits the same greenish discolouration. You can only see it by standing directly before the patient, not by looking at the eye sideways. This is called glaucoma simply, and it appears to me to be a chronic form of the same affection as to that to which the term acute glaucoma is given."[10]

In no writer at that period is the recognition of the identity between arthritic ophthalmitis and glaucoma to be found—the general conception being that glaucoma is a condition which may supervene on some cases of arthritic ophthalmitis. Lawrence saw further than this, and though the credit for introducing the term acute glaucoma must be given to him, it must be recognized that in linking up arthritic ophthalmitis and acute glaucoma he did so not on the basis of the modern conception of glaucoma, but on the elusive greenish discolouration.

It must further be admitted that in spite of his deep insight into the problem he did not grasp the whole position. In common with his contemporaries he considered most cases of what we now know as chronic glaucoma as nothing but amaurosis. The chronic glaucoma of Lawrence and his generation was what we would now consider absolute glaucoma following either an acute or chronic glaucoma. Not until the conception of ocular hypertension and the findings of the ophthalmoscope had been applied could glaucoma simplex become a clinical entity. This group was missed even by von Graefe[11] who in 1857 spoke of glaucoma of the acute, chronic and secondary types and of amaurosis with excavation of the disc. Not till Donders[12] (1861) was the unifying conception achieved.

The term acute glaucoma found its way into text books more rapidly than is generally the case with new names. It does not appear in the first edition of Mackenzie's classical book (1830)[13] though arthritic iritis and glaucoma are fully described. But it finds a place in the second edition, five years later, and in Richard Middlemore's Treatise[14] published in that same year.

Mackenzie's indebtedness to Lawrence is clearly brought out by a volume in possession of the Ophthalmological Society of the United Kingdom. It is a first edition of Mackenzie's book, interleaved and profusely marked with holograph notes. The book has

been fully described by R. R. James in an unsigned Annotation.[15] These notes are largely the additional matter of the second edition. One such note refers to acute glaucoma and is shown in the adjoining reproduction. In the second edition this particular marginal note does not appear, though another reference to acute glaucoma does appear for which no manuscript notes are present.

I am indebted to Mr. R. R. James for his information on the history of Mackenzie's book and its holograph notes.

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JUBILEE OF THE FRENCH OPHTHALMOLOGICAL SOCIETY

THE French Ophthalmological Society celebrated the 50th anniversary of its foundation in July and, for the occasion, a large number of guests were invited, representing nearly every country in Europe, and also Egypt and the United States. The guests who accepted the invitation were entertained at the Hotel Majestic where, during the Peace Conference, the British Delegates were lodged. The lounge of the Hotel during the meeting came to resemble a miniature International Congress, with representatives of nearly every European nationality. Among the guests were Professor and Mrs. Wagenmann from Heidelberg, and Professor

Krückmann from Berlin, Dr. and Mrs. Walter Parker (Detroit), Mr. and Mrs. Leslie Paton (London), Mr. and Mrs. Adams (Oxford), Professor Sachs (Vienna), Professor and Mrs. van Duyse (Ghent), Professor Pascheff (Sofia), Docteur Vogby (Giza, Egypt), Professor and Mrs. Marquez, Dr. and Mrs. Lloveras, and Dr. and Mrs. Marin Amat (Madrid), Professor and Mrs. Weve (Utrecht), Professor and Mrs. van der Hoeve (Leiden), Dr. and Mrs. Marx (Rotterdam), Professor de Grósz (Buda Pesth), Professor and Mrs. Ovio (Rome), Dr. and Mrs. Lodato (Palermo), Dr. Slavick (Tscheko Slovakie), Dr. Manolesco (Bucharest), Professor Szymanski (Warsaw), Dr. and Mrs. Patry (Geneva) and Dr. and Mrs. Pflüger (Berne). The majority of the guests arrived on Sunday, July 17, and the meetings commenced on Monday, July 18. The mornings were devoted to the more strictly scientific work of the Congress and as the meetings commenced at 8·30, it allowed over four hours daily for the reading and discussion of papers. In all, between 70 and 80 papers were read and many of them gave rise to useful discussions. The papers on Monday mostly dealt with diseases of the lids and anterior portions of the eye, many of them illustrated. On Tuesday morning the general discussion on "The Vitreous" took place. It is the custom of the French Society to choose a subject for discussion two years ahead and to ask one or two members of the Society to prepare reports on the subject which are published some months before the meeting at which they are to be discussed. Last year Professor van Duyse of Ghent prepared a valuable monograph on "Heredity in Eye Disease". This year, Redslob of Strasburg and Koby of Basle were the two reporters and their reports appeared some months ago, beautifully illustrated. Redslob's title was "Le Corps Vitré, son developpement, sa structure, ses propriétés physicochimique." Koby dealt with "Biomicroscopie du Corps Vitré." In addition, there were papers by Dejean and Mawas on the same subject and a very interesting discussion followed. Wednesday's papers were mainly on retinal subjects and, as was to be expected, retinal detachment bulked largely in the programme. Out of a total of 17 papers, 9 were devoted directly to this subject. It was very pleasing to hear an excellent paper from L. Coppez, representing the third generation of a distinguished family of Belgian ophthalmologists. Thursday's programme was of a more mixed character. Amongst the readers were Bailliart, Terson, Onfray, Coppez, and Marquez, names all well known in England for good work. Coppez' cinematographs of synergic ocular movements were specially interesting.

It is one of the pleasant habits of the French Society to ask their foreign guests to preside over the meetings and the Chair was occupied on various occasions by a Spaniard, Marquez (who will preside over the International Congress at Madrid next year), a

German, Krückmann of Berlin; an Austrian, Sachs of Vienna; a Hungarian, de Grósz of Buda Pesth; an Italian, Ovio of Rome; a Sicilian, Lodato of Palermo; a Dutchman, van der Hoeve of Leiden, and others. Nothing could show better the catholicity of the Society than the above selection.

The afternoons and evenings were given up to meetings of a more social nature. The presentation of addresses of congratulation was made at a special meeting on Monday afternoon where the Chair was taken by His Excellency, the Minister of Public Health, M. Justin Godard. Paton, of London, presented an illuminated address on behalf of the Ophthalmological Society of the United Kingdom, which was received with acclamation. It was in the followng terms:—

"Gallorum Societati Ophthalmicorum quinquagesimum annum aetatis suae hodie celebranti Britannorum Societas fraterna salutem nuntiat.

Cum labore, disciplina, doctrina summam ubique laudem merito sitis consecuti gratulamur vobis. Quod tot milibus lucem salutemque reddidit ac restituit ars vestra, quod tantis beneficiis totum orbem terrarum scientia vestra cumulavit, laetamur et ex animo gratias vobis agimus."

The main addresses from other countries were presented by Krückmann from Germany, Sachs from Austria, van Duyse from Belgium, Parker from America, Marquez from Spain, Weve from Holland, de Grósz from Hungary, Ovio from Italy, Patry from Switzerland, Szymanski from Poland, and representatives from nearly all the other countries of Europe. van der Hoeve spoke on behalf of the International Ophthalmological Council.

René Onfray, the energetic Secretary-General, to whom the Society owes the very greatest debt of gratitude, gave a most eloquent and interesting account of the birth and history of the Society, which, it is to be hoped, will be published in full.

Later on Monday afternoon, the Paris Municipal authorities held a reception to the members of the Society in the magnificent hall of the Hôtel de Ville, and in the evening, there was a Banquet at the Hôtel Majestic, presided over by M. Justin Godard. On this occasion, Professor van der Hoeve took the opportunity of presenting, on behalf of the invited guests of the Society, a silver Chairman's bell, beautifully modelled and engraved.

On Tuesday afternoon, the main social function was an excursion by river to Sèvres and a visit to the State factory of porcelain where everything was done to allow the visitors to see the whole process of manufacture and decoration of the famous ware. Leaving Sèvres, the members went on to St. Cloud where they dined at the Pavillon Bleu.

On Wednesday afternoon, Dr. Mawas, President of the Ophthalmological Society of Paris, entertained the members at a Thé Dansant in the salons of the "Cercle de l'Union Interalliée," familiar to many British officers as the house presented by M. Henri de Rothschild to form an inter-allied officers' club during the War.

A performance at the Opera House of "L'Illustre Frégona," with its cheerful exhibition of various Spanish dances, formed a fitting climax on Wednesday evening to a most charming series of entertainments.

It would be impossible to refer to all the hospitality shown in private by members of the Society resident in Paris to their guests, but to all who were fortunate enough to be invited, these parties will be among the most pleasant memories of a very memorable week, which gave all who took part in it the opportunity of renewing old friendships and forming new ones.

We who were the guests of the Society can only express our most sincere thanks to the Society and especially to its most amiable Secretary, Dr. Onfray, for their most generous hospitality to us on the occasion of their "Cinquantenaire."

BRITISH MEDICAL ASSOCIATION

Centenary Meeting

THE Ophthalmic Section of the British Medical Association Centenary Meeting met in London on July 27 and 28, 1932, under the Presidency of Sir John Parsons.

The morning of July 27 was devoted to a discussion on ocular manifestations of lesions of the Fifth Nerve.

Mr. FOSTER MOORE in his opening paper referred to the close relationship between Herpes Zoster Ophthalmicus and Varicella, and described the pathological changes found in the Gasserian ganglion in the former condition. The acute inflammatory changes in the ganglion are more pronounced if the eruption is severe, leading to permanent loss of function associated clinically with neuro-paralytic keratitis. In mild cases recovery is complete. Herpes zoster might occur as early as four years of age, but the average age was 41 to 55 years. General paralysis and tabes were predisposing causes. Intra-ocular complications occur in 60 per cent. of cases and in these cases usually the naso-ciliary nerve is affected, with a rash on the side of the nose.

Associated ocular palsies usually cleared up in time. The third nerve was affected in 74 per cent. of these cases. Optic atrophy sometimes occurred, and non-ulcerating nodules were occasionally found in the sclera which left brown spots at which the conjunctiva is adherent to the sclera.

As an alternative to the theory of anti-dromic influences, he referred to the possibility of the products of disintegration of ganglion cells passing down by the nerves to the skin. Mr. Foster Moore believed in tarsorrhaphy for neuro-paralytic keratitis and for conditions where the ganglion was destroyed.

Dr. C. M. HINDS HOWELL said that spontaneous corneal ulceration was most likely to occur when the first neurone was affected in its extra-medullary portion. He had never seen it develop in supranuclear or medullary lesions. Surgical methods of treating tic douloureux showed an advantage over alcohol injections, in that it was possible to avoid damage to the fibres of the first division of the fifth, which is rarely affected primarily.

Of 60 cases treated by alcohol injection, 36 had complete anaesthesia of the first division and 16 of these developed corneal ulcer. The first fortnight was the most dangerous period for the cornea. He remarked on the increased effect of X-Rays on insensitive tissues, and said that malignant tumours appear to advance more rapidly if the sensory nerve supply to the part affected is cut off.

Mr. HUGH W. B. CAIRNS discussed lesions of the fifth nerve from the surgical point of view, expressing a preference for post-ganglionic neurotomy.

An interesting discussion followed in which Dr. HUGH CARLETON, Mr. DOGGART, Mr. BALLANTYNE, Mr. CHARLES RUDD, and others joined. Dr. RUSSELL BRAIN referred to the possibilities of treatment in cases of Herpes zoster; intra-thecal administration of sodium iodide in the acute stage, pituitrin in a later stage, and X-Ray therapy in the second or third week.

Thursday morning was devoted to short papers.

A very interesting paper was read by Squadron-Leader P. C. LIVINGSTONE, R.A.F.M.S., on "Some Effects of Glare upon the Eyes." He gave the results of a large number of careful observations he had carried out on patients in the neighbourhood of Baghdad where there is much glare. He found that those who had been exposed to the glare for prolonged periods showed certain changes in the visual fields, including large scotomata, which were not present in newcomers to the country.

Mr. BISHOP HARMAN demonstrated a "Projection Scotometer" in which the usual test object is replaced by a spot of light projected from the instrument on to a white or grey surface.

On Wednesday afternoon a *scientific demonstration* was arranged at University College, in association with the Physiological

Society. The demonstration was almost unique in that so many of those engaged in laboratory research on subjects of Ophthalmological interest, were present to demonstrate their work.

The following is a brief account of the various demonstrations.

Mr. W. S. STILES showed a Photometric Pupillometer, an apparatus for determining the diameter of the pupil based on matching a photometric field. Also a "Brightness difference threshold meter," an apparatus for measuring the deleterious effect of glare on visibility.

Mr. J. GUILD demonstrated an apparatus showing the visual acuity attainable at very high field brightness. A miniature set of Snellen's Letters engraved on glass, was illuminated by transmitted light. These letters could still be read when the intensity of the light was increased to such a degree that a central scotoma persisted for several minutes after making the observation.

Professor POPA and Dr. FIELDING demonstrated the portal blood supply of the pituitary gland, while anatomical demonstrations of the orbit and cranial nerves were shown by Professor H. H. WOOLLARD and Mr. E. WOLFF. Professor H. A. HARRIS showed various specimens of historical interest relating to localization in the cerebral cortex, and gave a demonstration on the comparative anatomical aspects of the visual apparatus.

Dr. J .H. SHAXBY showed a "Two Collimator grating Spectrometer," useful for superimposing spectral colours in the examination of colour defective subjects and for other purposes.

He also showed a simple pocket photometer for measuring the reflecting power of the coal face in a mine, or for observations on the skin in anthropometric or clinical work.

Miss IDA MANN demonstrated clinical methods of examination applied to the eyes of living animals, emphazising certain genetic characters in typical eyes of mammals.

Her exhibits were arranged under three headings :—

1 A series of animals illustrating six of the commoner types of fundi. Orange red fundus (man and the primates); iridescent green and brown fundus with tapetum (cat); amphibian fundus with visible circulation in the retinal vessels (African nirmskull frog); ophidian fundus with tunica vasculosa retinae (Corais snake); lacertilian fundus with simple conical pecten (Callopistses-yellow spotted lizard); avian fundus with plicated pecten (pigeon).

2 A series of animals showing modifications of the lids.

Nictitating membrane (opaque in the pigeon, transparent in the crocodile).

Transparent lid (frog and skink).

Closing eye by raising lower lid (frog and lizard).

Permanently closed lids with transparent corneal scale (snake).

Lids almost completely fused so that only pupil is exposed (chamelion).

3 The slit lamp appearances of four types of vertebrate iris.

Mammalian arrangement (albino guinea-pig).

Elaboration of sphincteric plexus in birds (pigeon).

Colour pattern continued from head into iris stroma (python).

Pigmented vessels and specialized pupil (gecko).

Mrs. CAMPBELL and Mrs. M. C. BOURNE demonstrated ocular lesions in rabbits following adminstration of naphthalene. These consisted of retinal exudates, lental opacities, and deposits (of crystals of calcium oxalate) in the retina.

The type of lesion produced is markedly affected by the diet. On a calcium rich diet, such as oats and cabbage, the lens is not affected, retinal exudate is scanty, but crystals are deposited in the retina. On a calcium poor diet, such as bran and carrots, retinal exudate is extensive and cataract develops.

One of the rabbits exhibited a remarkable deposit of glistening crystals in the retina.

Professor E. D. ADRIAN gave a demonstration of the discharge of sensory impulses in the optic nerve when the eye is exposed to light, and the audience had the novel experience of listening to the sensory messages passing from the eye to the brain.

The eye of a water beetle was set up in a dark chamber with electrodes leading from the optic ganglion and the optic nerve. The electrodes were connected with an amplifier and a loud speaker so that the passage of a nerve impulse with its accompanying electric discharge gave an audible click, and a discharge of impulse in the whole nerve gave a loud noise. Feeble illumination of the eye produced an immediate discharge in the optic nerve, and the changes in the intensity of the sound showed how the discharge varied with dark adaptation, moving or steady light, etc.

Oscillograph records of the nerve impulses were also shown to illustrate the general use of the method in the investigation of the sense organs.

Mrs. LYTHGOE (*nèe* TANSLEY) demonstrated the development of visual purple in the rat retina. It was shown that the rat retina is not fully developed until 20 days after birth, and that visual purple does not appear until the rods have got well developed outer limbs. The development of the rat retina *in vitro* was also demonstrated by living cultures and stained specimens, showing that the immature retina can be made to develop rods and cones after removal from the body by cultivation in artificial media. It was also shown that in tissue cultures of the retina, rosettes are formed which closely resemble those found in glioma retinae.

Professor H. E. ROAF demonstrated some points in connection with colour vision. He showed how a sensation of yellow is

produced by binocular combination of red and green, and how a sensation of blue is produced by "secondary excitation" when observing a spot adjacent to a small point of red or green light. He demonstrated the poor visual acuity obtained in blue light, suggesting that the receptors for blue represent a larger area than those for red and green. He also showed how a coloured diagram and a copy made by a hypochromat look alike if viewed through a blue filter, but if viewed through a red or green filter the mistakes stand out by contrast. He demonstrated the interference with the visibility of one coloured light by another light, and showed the presence of red, yellow, and colourless globules in the cones in the retina of a bird.

Mr. R. J. LYTHGOE showed an apparatus for photographing the pupil in red light. The source of light was an arc light focused through an orange-red filter on the eye. The light was flashed on for 1/30th of a second and the photograph taken on very rapid panchromatic plates. He also showed a simple apparatus for recording small quantities of light by means of bromide paper standardized by comparison with similar paper exposed behind a neutral tint wedge.

He also demonstrated the Pulfrich phenomenon, showing how the swing of a pendulum appears to take the form of an ellipse if viewed binocularly with a dark glass before one eye. This is due to the delayed reaction time of the darkened image and the displacement is proportional to the logarithm of the ratio of the brightnesses of the two retinal images. A similar effect is produced if one eye is subjected to glare.

Mr. W. S. DUKE-ELDER and Mr. E. B. ROBERTSON demonstrated the ultra-microscopic appearances of the vitreous body and blood plasma. The substance to be examined was placed in a black glass container illuminated by the focused light of a carbon arc. The ultra-microscope was arranged at right angles to the beam of light so that the light entering the microscope is scattered from the particles in the medium. The vitreous viewed in this manner showed a structure of fine interlacing fibrillae which persist under sterile conditions for some ten days. A similar appearance is given by 1 in 50 horse plasma in normal saline.

The fibrillae in the first case probably consist of "residual protein," in the second case, of fibrin.

They also demonstrated a magnetic method for the measurement of the rigidity of the vitreous body. The vitreous was contained in a glass cell, and a small (20μ—200μ) particle of nickel drawn into the gel by means of a powerful magnetic field. The nickel can then be deflected by means of a suitable electro-magnet, its movements being observed and measured through a microscope. The movements are rapid at first and become slower, corresponding to those obtained for gelatine gels or plasma.

Professor H. HARTRIDGE demonstrated chromatic aberration in the eye and an apparatus permitting direct measurements; also a quantitative test for colour blindness.

Mr. J. W. TUDOR THOMAS demonstrated rabbits with transparent corneal grafts. One of these grafts had been observed for three years and four months and still remained transparent.

A graft of sclera on to the cornea of a rabbit was also shown.

J. W. TUDOR THOMAS.

ABSTRACTS

I.—LENS

(1) **Cole, Leslie (Cambridge).—Parathyroid tetany and cataract.** *Lancet*, January 4, 1930.

(1) **Cole's** article is mainly concerned with the detailed history of a case of tetany in which this condition, and eventually cataract, developed as the result of surgical removal of the two lateral lobes of the thyroid. Treatment with parahormone had no effect on the cataracts which, if one rightly understands the author, were being operated on by Mr. Arthur Cooke by needling, at the time of writing. The patient is aged 34 years. The author refers very briefly (at the commencement of his article) to the literature, beginning with Erdheim in 1906, showing the association of cataract and muscular spasms with parathyroid deficiency.

ERNEST THOMSON.

(2) **Branbergen, R. T. (Dordrecht).—Cataract after thyroidectomy. (Cataracte après Thyroïdectomie).** *Arch. d'Ophtal.*, February, 1931.

(2) This is the record of a case in which **Branbergen** watched the development of bilateral cataract as a sequel to thyroidectomy. A married woman, aged 34 years, underwent thyroidectomy in June, 1924, local anaesthesia being employed. The thyroid gland was firmly adherent to surrounding tissues: the median portion was completely removed: the wound healed normally.

The following day widespread "spasms" developed, which were treated and controlled by calcium. (The author does not use the term *tetany* in his paper).

Four years later vision in both eyes began to fail; after a year of

almost stationary defect rapid deterioration ensued, especially in the left eye.

In the early part of 1929, the patient was liable to occasional attacks of spasm. The hair and nails showed no degeneration, and menstruation continued normally.

Examination of the eyes revealed no abnormality except in the crystalline lenses. With the slit-lamp was visible immediately beneath the capsule a thin layer of degeneration, more delicate under the anterior capsule. It was composed of very fine elements assuming the form of snowflakes, between which were minute coloured particles of crystalline appearance.

Visual acuity quickly fell; early in 1930 it was less than 1/10 in each eye. The left cataract was extracted with resulting vision of 3/4.

Cataract as a sequel of removal of the thyroid gland is comparatively rare; most of the recorded cases have occurred in Switzerland. Jacques, in 1928, in an extensive search, collected 34 examples in the literature. It is only in recent years that biomicroscopy has furnished a precise description of the form of cataract, but there appears to be considerable variation in the character of the lental changes.

In the cases quoted by Branbergen, there is a record of tetany or severe spasm in the majority, leading in some to a fatal result. Organo-therapy is usually employed when tetany ensues; it is not always effective, and, as in the author's case, the administration of calcium may be more successful.

J. B. LAWFORD.

(3) **Leech, Vernon Mayne (Chicago). — Persistent posterior fibrovascular sheath of the lens caused by foetal iritis.** *Arch. of Ophthal.*, September, 1930.

(3) **Leech's** case is interesting in that the persistence of the posterior fibrovascular sheath of the lens was associated with definite histological signs of inflammation. The patient was a girl, aged four months, in whom the right eye was removed for suspected glioma. The inflammatory signs comprised (1) Cells in the anterior chamber; (2) Fibrosis of the iris with cellular infiltration; (3) Capsular cataract and shrinking of the lens; (4) Retention of the pupillary membrane and posterior fibrovascular sheath with its attached hyaloid artery. This latter is regarded by the author as an attempt by nature to combat the organisms causing the inflammation, by retaining the rich vascular network which normally disappears. He suggests that arrested development of other eyes may be due to a toxaemia of some sort.

F. A. W.-N.

(4) **Kirby, Daniel D. (New York).—Exfoliation of the most superficial lamella of the anterior capsule of the crystalline lens.** *Arch. of Ophthal.*, July, 1930.

(4) **Kirby** records seven examples of this condition. The characteristics are, the appearance of a very roughened surface on the capsule of the lens, with light bluish-grey opaque flakes. These flakes may also be present on the iris and the posterior surface of the cornea. There is usually a ring of capsule tags adherent to the lens in the area of maximal contraction of the pupil and the appearances suggest that the movements of the iris have denuded the degenerate capsule of its superficial layers. In confirmation of this is the observation that in all his cases Kirby found an associated senile change in the cortex of the lens. Some of the particles of capsule may be liberated into the aqueous and cause blocking of the pectinate ligament, thus bringing about glaucoma. This complication occurred in 75 per cent. of a series of 45 patients observed by Vogt. This observer examined one eye histologically, post-mortem, and found a definite separation of the superficial lamella of the capsule.

F. A. W-N.

II.—REFRACTION

(1) **Kazdan, Louis (Toronto).—The use of contact lenses for the optical correction of conical cornea.** *The Canadian Med. Assoc. Jl.*, December, 1931.

(1) In the opening sentences of this article the writer gives some historical facts concerning contact lenses. No genuine success in their manufacture or use was obtained until the Zeiss optical works, at Jena, produced the contact glasses now generally employed. **Kazdan** gives a description of such glasses, but as these are now familiar to most ophthalmologists this need not be repeated. They are made from the hardest optical glass and are, or should be, quite resistant to the corrosive effect of the tear fluids.

With the test contact glass in position in the patient's eye, ordinary lenses are added until the maximum vision is obtained. The strength of the lens, plus or minus, is then incorporated in the contact lens prescribed. "It requires time and patience on the part of both oculist and patient, until the latter becomes accustomed to insert and remove the contact glass himself. At first the eye is anaesthetised . . . but after several trials the use of anaesthesia can be discontinued." The author advises that a contact glass should not be worn continuously for more than about eight hours.

The apparatus he recommends for insertion is described and illustrated.

Kazdan's two cases, (*a*) a male, aged 44 years, whose sight failed in early adolescence, and (*b*) a female, aged 38 years, in whom conical cornea developed at the termination of her school career, were greatly benefited by the use of contact lenses.

(*a*) V. was in R. about 7/200, L. about 4/200. Left eye was divergent. With contact glasses and added correction vision was improved to R. 20/30, L. 20/40. Owing to the strabismus, correction to the right eye only was prescribed.

(*b*) A school teacher: V. was 1/200, R. and L. With contact lenses vision was improved to 20/200, and "4 mm. print."

"Both these patients have for the first time since the onset of their disease been able to see their own features in the mirror, and to see the world about them as it really is."

A bibliographical list is appended.

J. B. LAWFORD.

(2) **Cattaneo, D. (Rome).—The alterations of the vitreous in myopia. (Le alterazioni del Vitreo nella Miopia).** *Boll. d'Ocul.*, April, 1931.

(2) **Cattaneo** has examined a number of myopes by means of the slit-lamp and the microscope and has found that changes in the vitreous are much earlier in appearance than in normal eyes.

Often there can be seen filiform masses floating in the fluid vitreous; Cattaneo does not agree with those who hold these always to be the remains of the hyaloid vessels; on the contrary he considers many to be due to the entrance of heterogeneous elements, and explains in this way the presence of changes in the lens and other structures. He finds that pigmented opacities are constantly found with detachment of the retina, and may be a forerunner of this condition.

HAROLD GRIMSDALE.

(3) **Blatt, N. (Tg. Mures).—Cyclic weakness of accommodation in dysmenorrhoea. (Zyklische Akkommodationsschwäche bei Dysmenorrhoe).** *Zeitschr. f. Augenheilk.*, Vol. LXXIII, p. 106, 1931.

(3) **Blatt** reports a case of a frail-looking but healthy girl, aged 19 years, in whom disturbances of accommodation recurred at each menstrual period. Normally menstruation lasted 8 days and was associated with general malaise in addition to backache. Accommodation was normal in the intermenstrual periods but within a day or two of the onset of menstruation the near point had receded from 10 cm. to 26-30 cm. Difficulty with near work was most marked towards the end of the day; in the morning reading was

possible for a short time, but in the afternoon it could not be done at all. Accommodation recovered gradually whilst the periods still lasted, so that by the end of menstruation it was practically normal. During the height of accommodative disturbances, attempts at reading rapidly exhausted what accommodation was available, such accommodation re-appearing after a short interval of rest. Possible explanations are discussed, and the author is inclined to regard these disturbances as due to auto-toxaemia acting on a system predisposed owing to endocrine disturbances.

ARNOLD SORSBY.

BOOK NOTICE

Das menschliche Auge und seine Erbanlagen. By Dr. P. J. WAARDENBERG, Ophthalmic Surgeon in Arnhem. With 8 coloured tables, and 196 illustrations in the text. Pp. XVI and 631. The Hague: Martinus Nijhoff. Price, 30 Dutch gld. 1932.

This book forms Volume VII of the Bibliographia Genetica, and is the most comprehensive study of hereditary diseases of the eye which has appeared. We in England are all familiar with the epoch-making researches of Nettleship on this subject; and these in turn have inspired the work of Usher, Julia Bell, Lawford and others. Unfortunately many of their communications are enshrined in special numbers of *Biometrica*, a costly production of limited circulation. Moreover, many as are the diseases so exhaustively investigated by these writers, they do not provide a complete conspectus of the whole subject as does the volume under review. Dr. Waardenberg chose hereditary disease as the topic for his "Inaugural Dissertation" in 1912, and has continued to collect material, particularly of eye diseases, ever since that date. The result is a masterpiece of industry, admirably arranged, and documented with full bibliographies. It cannot be neglected by any student of heredity, *a fortiori* by any student of inherited affections of the eyes. Apart from the encyclopaedic material which forms the body of the book the attention of ophthalmologists may be specially directed to the thoughtful discussion of the true criteria of inherited disease contained in the short introduction.

The author first discusses variations of pigmentation; then passes on to consider each structure of the eye and its adnexa seriatim; concluding with chapters on the eye and nervous system, refraction, variability and asymmetry, twins, etc. These chapters are a rich storehouse of data.

The book is beautifully printed and illustrated.

OBITUARY

JOHN FRANCIS CUNNINGHAM, F.R.C.S., O.B.E.

J. F. CUNNINGHAM, whose death at the early age of 56 years, we record with regret, was the elder son of Mr. J. K. Cunningham, of Sec Tor Hall, Axminster. He was educated at Sherborne School and St. Thomas's Hospital. After obtaining the diploma of the Conjoint Colleges in 1900, he filled the offices of House Surgeon, Ophthalmic House Surgeon and Ophthalmic Registrar to St. Thomas's Hospital, and Demonstrator of Physiology in its Medical School. He was also House Physician to Bethlem Royal Hospital.

Subsequently he became a student at Moorfields Hospital, where for several years he did good work as Chief Clinical Assistant. He obtained the F.R.C.S. in 1905.

In 1910 Cunningham was elected to the staff of the Central London Ophthalmic Hospital, and to its service and interests he gave devoted and unremitting attention until the onset of his fatal illness. In addition he held the posts of Ophthalmic Surgeon to St. Charles's Hospital and Ophthalmologist to Bethlem Royal Hospital. He was a member of the Ophthalmological Society and served as Secretary in 1922-1924. He was also Secretary to the Section of Ophthalmology of the R.S.M.

During the War, Cunningham was in France from March, 1915, until December, 1918, serving in the R.A.M.C. with the rank of Major. He was mentioned in dispatches and received the O.B.E.

In his professional work Cunningham was very thorough and painstaking, never grudging time or effort on behalf of those under his care. His personal character was very attractive; his obvious straightforwardness and genuine sincerity endeared him to a large circle of friends.

He married in 1910 a daughter of the late Mr. Justice Clarence of the Supreme Court of Ceylon, and had two sons and three daughters.

By one who knew him during the War.

The stimulus of the War, in developing the desire to serve, accentuated an indigenous characteristic in Frank Cunningham. Directly the War began it was his great aim to go abroad, and as soon as the opportunity arose he came to France with alacrity. For four years he worked there with tireless enthusiasm for the British Expeditionary Force.

By nature somewhat retiring, diffident in pressing his opinions,

and entirely antagonistic to all self-seeking and advertisement, he was so patently genuine and out to help in every way in his power that he completely won a position of distinction by his good judgment and meticulous thoroughness, and proved himself the ideally loyal colleague.

His previous experience in trachoma at special centres in England made his assistance invaluable in the difficult problem of dealing with the many thousands of Chinese and Egyptians recruited in the Labour Corps, who were found to be suffering from this intractable disease. In addition his wise suggestions as well as his tactful dealing with the authorites were very material in the successful handling of the situation.

Throughout his period of War Service he endeared himself to all who worked with him, and for whom he worked, by his unselfishness and cheery kindliness. His loss will be felt deeply by many.

FUTURE ARRANGEMENTS

1932

October 4.—Midland Ophthalmological Society, at Birmingham Eye Hospital.

CONTEMPORARY OPHTHALMIC LITERATURE

American Journal of Ophthalmology. *July, 1932.*

SPAETH. Some biological principles which underlie ophthalmic plastic surgery.
LUEDDE. Monocular cycloplegia for the control of myopia.
DANIELSON. Some theoretical and practical points regarding unilateral voluntary winking.
WOLFE. Notes on muscle and fusion training and orthoptic treatment of motor anomalies.
OLÁH. The technique of preparing for cataract operation.
FERREE and RAND. A convenient device for using artifical pupils of different sizes.

Archives of Ophthalmology. *June, 1932.*

BLAIR, BROWN and HAMM. Correction of ptosis and of epicanthus.
RONES. Uveitis with dysacousia, alopecia and poliosis.
KLAUDER and DELONG. Lupus erythematosus of the conjunctiva, eyelids and lid margins.
SAMUELS. Some notes on orbital tumors.
MAYER. The vitreous in experimental detachment of the retina.
ABRAHAM. Anterior chamber punctures in relation to intra-ocular tension.

RABKIN. Post-operative care without bandages.
AMES and OGLE. Size and shape of ocular images. III. Visual sensitivity to differences in the relative size of the ocular images of the two eyes.

July, 1932.

LANCASTER. The ophthalmologist and the lighting problems of his patients.
FRIEDENWALD and PIERCE. Circulation of the aqueous. II. Mechanism of reabsorption of fluid.
LOURIE. Abuse of the theory of focal infection.
FERREE and RAND. Lighting without glare.
COHEN, KILLIAN and HALPERN. Chemical composition of the blood and spinal fluid in primary glaucoma. Comparative studies.
PASCAL. Simple method for calculating dioptric power.
MEYER. Mechanism of accommodation studied experimentally.
MANN. Grouped pigmentation fo the retina.
KRUG and ROHDENBURG. Foreign substances injected into the vitreous of the rabbit. Pathway of transportation from one eye to its fellow. An experimental study.

Annales d'Oculistique. *June, 1932.*

REDSLOB. True and apparent anophthalmia in an infant.
PATRY. Cavernous angioma of the lacrimal gland.
RISCHARD. The motor innervation of the iris.
LAFON. School optometric scale.

July, 1932.

REDSLOB. Contribution to the study of the pathogenesis of retinitis proliferans.
LEPLAT. Contribution to the study of the vitreous body.
BUJADOUX. Collected reports on 44 cases of cataract operation by the method of Junès

Archives d'Ophtalmologie. *July, 1932.*

MARX and BOEVÉ. Meningiomata of the sella turcica.
WEEKERS. Treatment of spasmodic entropion by injection of the lid with alcohol associated with canthotomy.
FRENKEL. On the annular contusion cataract of Vossius.
LAMBERT. Contribution to the study of contusions of the eye in military service.
DEJEAN. On the possible rôle of polypeptidaemia in the genesis of renal retinitis.
RABINOWITSCH. Contribution to the study of transitory diplopia.
DELORD. Syndrome of the internal edge of the cerebral peduncle.
FAVORY. On a rare ocular complication of scarlet fever.
——— The prevention of blindness in India.

August, 1932.

WEEKERS. A new clinical sign for the differential diagnosis and prognosis of retrobulbar neuritis.
PASCHEFF. Researches on the diffuse lymphamotoses of the conjunctiva.
KARBOWSKI. A new modification of treatment in obliteration of the naso-lacrimal canal.

Revue Internationale du Trachome. *July, 1932.*

TALBOT. Prophylaxis of pandemic trachoma.
MACCALLAN. Cases of trachoma in English schools.
BONNET. Note on school trachoma in Algeria.
SÉDAN. Trachoma in tuberculous patients.
ROSTKOWSKI. Organisation of the fight against trachoma in Vilno and some clinical observations.

Archiv für Ophthalmologie. *July, 1932.*

PILLAT. The origin and object of the pigmentation of the conjunctiva in avitaminosis of adults and on the connection between avitaminosis and the suprarenals.
RÖTTH. On the aetiology of trachoma.
GOLDMANN. Experimental investigations on the genesis of heat cataract.
SCHMELZER. Experimental studies and clinical observations with Besredka's anti-virus.

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VELHAGEN, JUN. Clinical meaning of atypical retinitis pigmentosa.
VOM HOFE. The reaction of the cornea to a single injection of foreign serum in rabbits with different stages of syphilis.
OLLENDORF. On the value of the erythrocyte sedimentation reaction in the aetiology of iritis and irido-cyclitis.
WAAY. A case of cyclic oculo-motor paralysis.
NAKAMURA. On our knowledge of the successful treatment of exudative uveitis and of sympathetic ophthalmia with "AO."
ROLETT. On two cases of copying ink pencil injury to the eye.
JUHASZ-SCHÄFFER. A typical case of chronic progressive external ophthalmoplegia.
STREBEL. On desensitization treatment of hay fever with the Pollysat method and its results during the last ten years.
SALUS. Harada's disease.

Annali di Ottalmologia e Clinica Oculistica.

February, 1932.

IAMANDI and LAZARESCU. Contribution to the evolution and differentiation of corneal pannus, eczematous and trachomatous.
DENTI. Note on tuberculosis of the conjunctiva.
SANTONASTASO. Ocular changes in leprosy *(continued)*.
MURA. Echinococcal cyst of the orbit.

Archivio di Ottalmologia. *June, 1932.*

SALA. Pharmacological study of the action of armina on the eye.
FERRARI. The test of Aldrich and McClure and the function of the capillaries in idiopathic detachment of the retina.
SGROSSO. On post-operative sympathetic ophthalmitis.

July, 1932.

SGROSSO. On post-operative sympathetic ophthalmitis *(continuation and end)*.
CORDERO. On penetrating wounds of the eye observed in the years 1901-1930 at the R. Clinica Oculistica in Parma.

Bollettino d'Oculistica. *May, 1932.*

HORNIKER and SALOM. Ocular changes in tuberose sclerosis.
FAVALORO. On the diagnostic importance of exploratory puncture in neoplastic affections of the eye and orbit.
FOCOSI. On a case of mycotic concretion in the lacrimal sac.
LEONARDI. The extraction of non-magnetic foreign bodies in the anterior chamber.
FONTANA. Cytological researches on the blood of individuals affected with phlyctenular conjunctivitis.
MONTALTI. On melanosarcoma of the orbit.

June, 1932.

BRUNO. Observation of the development of the external layer of the optic cup and of the pigment.
SGROSSO. On an anomaly of development of the anterior uveal tract.
BELLAVIA. Action of chlorophyll on the ocular tone.
BARLETTA. On the behaviour of S.R.E. in experimental traumatic cataract.
TIERI. On keratoconus with special reference to surgical treatment.
DE VINCENTIIS. Cataract extraction with lateral conjunctival bridge.

Rassegna Italiana d'Ottalmologia. *March-April, 1932.*

VOLLARO. A personal symplified method to obtain relaxation and paresis of the lids in ophthalmic operations.
FAVALORO. On plastic operations in the naso-lacrimal region.
BARLETTA. The chiasma and the optic nerve in experimental beri-beri of pigeons.
MARCHESINI. The double palpebral eversion of Pes.
TIRELLI. On a case of abnormal length of the lacrimal canaliculi.
CORRADO. Hole at the macula of congenital origin.
GIANI. Traumatic angiopathy of the retina.
GALLENGA. On the behaviour of the pupillary diameter on high mountains in connection with fatigue.

Archivos de Oftalmologia Hispano-Americanos. *July, 1932.*

LLORCA. On a case of external exudative retinitis of Coats.
ARRUGA. Ocular diabetes.
FERRER. Treatment of ophthalmic tuberculosis with guayacol sulphonate and calcium.
AMAT and ENCISO. Sympathetic iridocyclitis cured by autohaemotherapy.

Boletin de Informacion Oftalmologica. *May-June, 1932.*

ONETO. On a case of hydatid cyst of the orbit.
DAMEL. Adherent leucoma in a trachomatous patient. Accident at work.
GALLINO. Free and moveable cyst in the anterior chamber.
MALBRAN. Ocular paralysis in spinal anaesthesia.
DAMEL. Hysteria in ophthalmology.

Revista Oto-Neuro-Oftalmologica y de Cirujia Neurologica Sud-Americana. *July, 1932.*

FUCHS. On ophthalmic "Jaqueca."
SPOTA. Sequel of injury to the basis cranii.
PAVIA. Congenital strabismus. Convergence or deorsumvergence?

August, 1932.

MENDOZA. Stereoscopic vision of space and relief.
PAVIA. The authentic recording of the fundus.
WEEKERS. Experimental ophthalmotonic reactions.

Revista Cubana de Oto-Neuro-Oftalmologica.

May and June, 1932.

LACARRERE. Our technique of cataract extraction with the electro diaphake.
FRENKEL. On annular contusion cataract of Vossius.
NICOLICH. What one should not forget in ocular antisepsis.